A DISSERTATION ON

"A COMPARATIVE STUDY OF LEVOBUPIVACAINE AND LEVOBUPIVACAINE WITH DEXMEDITOMIDINE IN USG GUIDED AXILLARY BLOCK FOR ELBOW, FOREARM & HAND SURGERIES"

Submitted to

THE TAMIL NADU DR. MGR. MEDICAL UNIVERSITY,

CHENNAI–600032. TAMILNADU.

In partial fulfillment of the regulations

For the award of the degree of

M.D. DEGREE BRANCH-X

ANAESTHESIOLOGY

GOVERNMENT MOHAN KUMARA MANGALAM

MEDICAL COLLEGE, SALEM, TAMILNADU.

APRIL 2015

Government Mohan Kumaramangalam

Medical College & Hospital

DECLARATION BY THE CANDIDATE

I hereby declare that this dissertation titled "A COMPARATIVE STUDY OF LEVOBUPIVACAINE AND LEVOBUPIVACAINE WITH DEXMEDITOMIDINE IN USG GUIDED AXILLARY BLOCK FOR ELBOW, FOREARM & HAND SURGERIES" is a bonafide and genuine research work carried out by me under the guidance of ***Dr. G. Sivakumar, M.D., DA., Professor***, Department of Anesthesiology, Government Mohan Kumaramangalam Medical College Hospital, Salem, Tamil Nadu, India.

Signature of the Candidate

DR.S.SYED THAHIR HUSSAIN.

Date: 14.10.2014

Place: Salem

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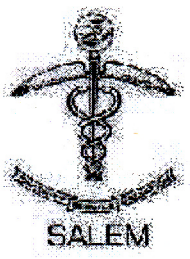

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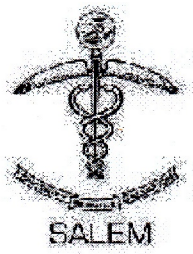

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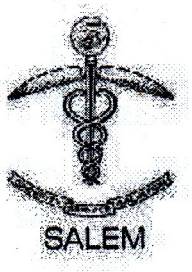

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Signature of the Candidate

DR.S.SYED THAHIR HUSSAIN.

Date: 14.10.2014

Place: Salem

ACKNOWLEDGEMENT

It is most appropriate that I begin by thanking the Almighty God for giving me the both mental and physical strength to accomplish this task.

I am extremely thankful to **Prof. Dr. N. MOHAN, MS.,** Dean, Govt. Mohan Kumaramangalam medical college & Hospital for allowing me to utilize the hospital facilities for doing this work, I am also thankful to **Prof. Dr. T. SWAMINATHAN, M.S.,** Medical Superintendent, Govt. Mohan Kumaramangalam Medical College & Hospital, for his wholehearted support and encouragement for the completion of this dissertation.

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I would like to express my deep gratitude and sincere thanks to my guide **Prof. Dr. G. SIVAKUMAR, MD., DA.**, for preparing me for this great task, guiding me with his excellent talent and professional experience, showing great care and attention to details and without his supervision and guidance, this dissertation would have been impossible.

I am highly indebted to all my **Assistant Professors** who taught me the ultrasound guided blocks and equip me with this skill.

I would like to express my deepest gratitude to my parents who prepared me for life and who led me to this run on ladder of my scholastic carrier, I am ever grateful to them.

I would also like thank surgeons and OT staff of GMKMCH, Salem for their help and assistance. I express my sincere thanks to post graduate colleagues and friends who have helped me in preparing this dissertation.

I am greatly indebted to all my patients for their co-operation in spite of pain and suffering from disease without whom this study would have been impossible.

Signature of the Candidate

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DATE:-14.10. 2014.

PLACE:-Salem.

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MINUTES OF THE MEETING.

Ethical Committee Meeting held on 06.12.2013 at 11.00 A.M.in the Dean's Chamber, Government Mohan Kumaramangalam Medical College, Salem 30.

The following Members were attended.

CHAIRMAN

Dr.A.Karthikeyan, MD., - Dean.

MEMBERS.

1. Dr.S.Mohammed Musthafa .M.D., - Vice Principal.
2. Dr.T.Swaminathan, M.S., - Medical Superintendent.
3. Dr.Priya Jayapal, M.D., HOD of Biochemistry.
4. Dr.R.Jayalalitha, M.D., HOD of Pharmacology.
5. Dr.M.Poovathi, M.D., HOD of O & G.
6. Dr.A.Ananthi, M.D., Associate Professor of O & G.
7. Dr.S.R.Subramanian, M.D.,HOD of Medicine.
8. Dr.R.Kattabomman, M.S., HOD of Surgery.
9. Dr.T.S.Sundararajan,M.D., HOD of Peadiatrics.
10. Dr.T.Sundararajan, M.D., Associate Professor of Microbiology.
11. Dr.C.Kamalanathan, M.S., HOD of Orthopeadics.
12. Thiru.J.M.Arumugam, Legal Advisor, Salem.
13. Tmt.Ruby Thiagarajan, Social Worker.

MINUTES OF MEETING.

The Vice Principal, Govt.Mohan Kumaramangalam Medical College, Salem -30 has welcomed all the members of the Committee and explained the purpose of the Ethical Committee Meeting and asked the applicants to present their study before the committee.

The following Post Graduate students of this College have presented their study and requested Ethical Committee clearance for submitting their Dissertation to the Tamil Nadu Dr.M.G.R.Medical University, Chennai.

DEPARTMENT OF SURGERY:

1. Dr.J.Prakash Kumar, IInd Year Post Graduate student of M.S.(General Surgery), Govt.Mohan Kumaramangalam Medical College, Salem has presented his Dissertation on " PROSPECTIVE STUDY OF COMPARATIVE STUDY OF CHEMICAL SPHGINCTEROTOMY (USING 2% DILTIAZEM) AND LATERAL INTERNAL SPHINCTEROTOMY FOR CHRONIC ANALFISSURE 100 CASES IN GMKMCH" under the guidance of Prof.Dr.P.Ramalingam, M.S, Associate Professor of General Surgery of this College.

2. Dr.S.Prasad, IInd Year Post Graduate student of M.S.(General Surgery), Govt.Mohan Kumaramangalam Medical College, Salem has presented his Dissertation on "A STUDY ON POSTOPERATIVE OUTCOME FOLLOWING TOTAL THYROIDECTOMY AND ITS COMPARISON WITH SUBTOTAL THYROIDECTGOMY IN MULTINODULAR GOITREDISEASE" under the guidance of Prof.Dr.V.Lakshmi Narayani, M.S, Professor of General Surgery of this College.

3. Dr.D.Jayaprakash, IInd Year Post Graduate student of M.S.(General Surgery), Govt.Mohan Kumaramangalam Medical College, Salem has presented his Dissertation on "A STUDY ON COMPARISION OF VARIOUS MODALITIES IN TREATMENT OF FISTULA IN ANO for a period of 2 years – IN GMKMCH, SALEM" under the guidance of Prof.Dr.C.Rajasekaran, M.S, Professor of General Surgery of this College.

3. Dr.S.Radhika, IInd Year Post Graduate student of M.D.(PAEDIATRICS), Govt.Mohan Kumaramangalam Medical College, Salem has presented her Dissertation on " A STUDY ON ETIOLGY, CLINICAL PROFILE AND OUTCOME OF NEONATAL THROMBOCYTOPENIA" under the guidance of Dr.P.Sampathkumar, M.D., Associate Professor of Paediatrics of this College.

DEPARTMENT OF ANAESTHESIA.

Dr.S.Syed Thahir Hussain, Ist Year Post Graduate student of M.D.(ANAESTHESIA.), Govt.Mohan Kumaramangalam Medical College, Salem has presented his Dissertation on "CLINICAL STUDY ON EFFECTS OF ADDING DEXMEDETOMIDINE TO LEVOBUPIVACAINE IN AXILLARY BRACHIAL PLEXUS BLOCK IN PATIENTS PRESENTING IN GMKMCH" under the guidance of Dr.K.Pandian,M.D., HOD of Anaesthesia of this College.

The Ethical Committee examined the protocol in detail and is pleased to accord Ethical Committee approval for all the above Post Graduate students of this College to carry out the studies and the committee also advised all the Post Graduate students to get Ethical Clearance before starting Dissertation work and they must submit the abstract and present their study along with their guides.

DEPARTMENT OF PHARMACOLOGY.

1. Dr.B.Keerthika, M.D., Assistant Professor of Pharmacology of this College has presented her study on "A PROSPECTIVE STUDY OF ADVERSE DRUG REACTIONS IN A TERTIARY CARE HOSPITAL" In-patient and

out patient departments of Medicine, Peadiatrics, Dermatology and Psychiatry if this College Hospital and requested Ethical clearance to carry out the study.

2. Dr.N.Preetha,M.D., Assistant Professor of Pharmacology of this College has presented her study on "A PROSPECTIVE ANALYSIS OF ANTIBIOTIC PRESCRIBING PATTERN AMONG IN-PATIENTS IN A TERTIARY CARE HOSPITAL" in the Hospital premises.

The Ethical Committee examined the study in detail and is pleased to accord Ethical Clearance for the above two Assistant Professors of Pharmacology to carry out the study without affecting their normal duties.

Further, the committee has disapproved the following study presented by the Post Graduate Students of this College.

1. Dr.G.Jasmine, IInd Year Post Graduate student of M.S.(General Surgery), Govt.Mohan Kumaramangalam Medical College, Salem has presented her Dissertation on " PROSPECTIVE STUDY OF 50 CASES ON ETIOLOGY AND PATHOLOGY OF CERTIVAL LYMPH NODES BY FNAC AND BIOPSY IN GMKMCH, SALEM" under the guidance of Prof.Dr.A.Nirmala.M.S, Associate Professor of General Surgery of this College.
2. Dr.Mohamed Yasid. IInd Year Post Graduate student of M.D.(General MEDICINE), Govt.Mohan Kumaramangalam Medical College, Salem has presented his Dissertation on " PULMONDRY FUNCTION TEST IN TYPO-Z DIABETES"

DEAN.

To

1. All the Post Graduate Students – Concerned through the HOD of respective departments.
2. Dr.B.Keerthika,M.D., Assistant Professor of Pharmacology of this College.
3. Dr.N.Preetha, M.D., Assistant Professor of Pharmacology of this College.

Copy to:

1. All the Members of the Committee.
2. The Vice Principal, Govt.Mohan Kumaramangalam Medical College, Salem 30.

Spare -1.

LIST OF ABBREVIATIONS USED

BP - Blood pressure

ECG - Electrocardiography

IV - Intravenous

LA – Local anesthesia

VAS- Visual Analog Scale

ORIF – Open reduction and internal fixation

- Fracture

MASTER CHART ABBREVIATIONS

HT- Hypertension

BB – Both bones

DM – Diabetes Mellitus

FA - Forearm

USG- Ultrasound

Deemed- Dexmeditomidine

Levo- Levobupivacaine

ABSTRACT

Background & Objectives:

The present clinical study was conducted to evaluate the onset of analgesia, degree of sensory and motor blockade, duration of analgesia and complications between Levobupivacaine and Levobupivacaine with Dexmeditomidine in USG guided axillary brachial plexus block.

Methods:

The study was conducted on 60 ASA 1 and 11 patients of either sex posted for various elective or emergency surgeries of the upper limb involving elbow, forearm and hand surgeries. The subjects were divided into two groups, group A receiving axillary brachial plexus block with 0.25% Levobupivacaine alone and group B receiving Levobupivacaine with Dexmeditomidine 0.5µg /kg. Patients' vital parameters were monitored throughout the procedure and in the post-operative period for 48 hours. A thorough observation was made on onset, quality, duration of analgesia, degree of motor blockade and complications.

Result

The onset of sensory blockade (mean difference 0.04 minutes, p-value <0.001) and motor blockade (mean difference 0.03 minutes, p-value <0.001) were quicker in group B compared to group A. Both these findings were statistically significant. Both the duration of sensory

blockade (mean difference 4.7 hours, p value <0.001), and motor blockade (mean difference 1.8 hours, p value <0.001) were longer in group B compared to group A and both these findings were statistically significant. Time taken for starting of regression (mean difference -1.37 minutes, p-value <0.108) was more in group B compared to group A, but this finding was not statistically different. All other parameters related to duration of anesthesia including time taken for full motor and sensory recovery were longer in group B compared to group A. These differences were statistically significant.

There were statistically significant differences in the duration of complete analgesia, duration of effective analgesia and time of first pain medication between the study groups. All these parameters were longer in group B compared to group A.

The average post anesthesia hemodynamic parameters like heart rate, systolic and diastolic pressures were higher in group A compared to group B. The difference in the heart rate and diastolic blood pressure were statistically significant. The post anaesthetic respiratory parameters were almost similar in both the study groups.

Interpretation and conclusion

In this prospective randomized double blinded case control study, Levobupivacaine with Dexmeditomidine seems to be advantageous over plain levobupivacaine in terms of onset, quality and intensity of sensory and motor blockade. The duration of analgesia is both clinically and statistically significantly prolonged in Levobupivacaine with Dexmeditomidine group.

Keywords: Levobupivacaine, Dexmeditomidine, USG, Axillary brachial plexus block.

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INTRODUCTION

The techniques of peripheral nerve blockade were developed early in the history of anesthesia. The US surgeons **Halsted and Hall** described the injection of cocaine in to peripheral sites which include the ulnar, musculo cutaneous, supra trochlear and infra orbital nerves for minor surgical procedures in the1880s.[1] **James Leonard Corning** recommended the use of an esmarch bandage in1885to arrest the local circulation; prolong the cocaine induced block, decrease uptake of that local anesthetic from tissues. This concept was further developed by **F.W.Braun** who substituted epinephrine, a chemical tourniquet in 1903. He also introduced the term conduction anesthesia in his 1914 textbook on Techniques of local anesthesia.

Peripheral nerve blockade remains a well-accepted component of comprehensive anesthetic care. Its role has expanded from the operating site into the arena of post-operative and chronic pain management. Skillful application of peripheral neural blockade broadens the anesthesiologists' range of options in providing optimal anesthetic care. The axillary approach to the brachial plexus is the most popular because of its ease, reliability and safety. Blockade occurs at the level of the terminal nerves and although blockade of musculocutaneous nerve

is not always produced with this approach, it can be supplemented at the level of axilla or at the elbow.

Indication:

Indication for axillary block includes surgery on the elbow, forearm and hand. This block is ideally suited for outpatients and is easily adapted to the pediatric population.

The introduction of percutaneous axillary and supraclavicular techniques by **Herschel** in 1911 was received enthusiastically during the First World War because of them any upper extremity injuries resulting from war. Brachial plexus block is widely used today to provide anesthesia for upper extremity. There are four usual sites of approach namely inter scalene, supraclavicular, infra clavicular & axillary.

Knowledge of formation of brachial plexus and its ultimate cutaneous and muscular distribution is absolutely essential for the intelligent and effective use of brachial plexus blockade for upper limb surgeries. Close familiarity with the vascular, muscular and fascial relationships of the plexus is equally essential to the mastery of various techniques; for it is these peri neural structures which serve as the landmark by which needle may accurately locate the plexus percutaneously. In its course from

intervertebral foramina to the upper arm, the fibers are composed consecutively of roots, trunks, divisions, cords and terminal nerves.

Formation of brachial plexus [2, 3]

Brachial plexus is formed by the union of ventral rami of lower four cervical nerves (C5, 6, 7, 8) and first thoracic nerve (T1) with frequent contributions from C4 or T2. When contribution from C4 is large and from T2 is lacking, the plexus appears to have a more cephaloid position and is termed **Pre fixed**. When contribution from T2 is large and from C4 is lacking, the plexus appears to have a caudal position and is termed **post fixed.** Usually pre fixed or post fixed positions are associated with the presence either of a cervical rib or of an anomalous first rib. (Fig.1)

Roots

Represent the anterior primary divisions of lower four cervical and first thoracic nerves. They emerge from the intervertebral foramina and fuse above the first rib to form the trunks.

Trunks

The roots combine above the first rib to form the three trunks of the plexus. C5 and C6 unite at the lateral border of the scalenus medius and form the "Upper trunk", C8 and T1 unite behind the scalenus anterior to

form "lower trunk" and C7 continues as a sole contributor to the "middle trunk".

Divisions

As the trunks pass over the first rib and under the clavicle, each one of them divides into anterior and posterior divisions.

Cords

The fibers, as they emerge from under the clavicle, recombine to form three cords. The "lateral cord" is formed by anterior divisions of upper and middle trunks, lateral to the axillary artery. The anterior division of lower trunk descends medial to the axillary artery forming the "medial cord". The posterior divisions of all three trunks unite to form the "posterior cord", at first above and then behind the axillary artery. The medial and lateral cords give rise to nerves that supply the flexor surface of upper extremity, while nerves arising from the posterior cord supply the extensor surface.[4] (Fig.2)

Major terminal nerves:

Each of these cords gives off a branch that contributes to or become one of the major nerves to the upper extremity and then terminates as a major nerve. The lateral and median cords gives off lateral and medial heads of the median nerve and continue as major terminal nerves, the lateral cord

terminating as musculo cutaneous nerve and medial cord as ulnar nerve. Posterior cord gives off, axillary nerve as its major branch and then continues as the radial nerve.

In summary, conveniently it can be considered that brachial plexus begins with five nerves (C5-T1) and terminates in five nerves (Musculo cutaneous, radial, axillary, median and ulnar nerves) with its intermediate portions displaying in sets of three that is, three main trunks which divide into 2 sets of three, which reunite and give rise to three cords. These three cords give off three lateral branches before becoming the major terminal branches of the plexus. (Fig.3 and 4)

Distribution of brachial plexus: [5.6]

These are divided into

A) Those that arise above the clavicle- the supraclavicular branches

and

B) Those that arise below it, the infraclavicular branches

Supraclavicular branches:-

From roots:

1. Nerves to scalenei and longus colli- C5.6, 7.8

2. Branch to phrenic nerve – C5

3. Dorsal scapular nerve – C5

4. Long thoracic nerve of Bell -C5, 6, (7)

From trunks:

1. Nerve to subclavius – C5, 6,

2. Supra scapular nerve-C5, 6

Infraclavicular branches:-

They branch from cords but their fibers may be traced back to spinal nerves.

Lateral cord:

1. Lateral pectoral nerve- C5, 6, 7

2. Musculo cutaneous nerve – C5, 6, 7

3. Lateral root of median nerve- C5, 6, 7

Medial cord:

1. Medial pectoral nerve- C8, T1

2. Medial cutaneous nerve of arm –C8, T1

3. Medial cutaneous nerve of forearm – C8, T1

4. Medial root of median nerve- C8, T1

5. Ulnar nerve- C7, 8, T1

Posterior cord:

1. Upper subscapular nerve-C5, 6

2. Lower subscapular nerve-C5, 6

3. Nerve to latissmusdorsi (Thoraco dorsal nerve) - C6, 7, 8

4. Axillary nerve-C5, 6,

5. Radial nerve- C5, 6, 7, 8, T1.

Supraclavicular branches:

As anesthesiologists, we must have complete knowledge of distribution of sensory fibers to upper extremities in order to provide surgical anesthesia appropriate for the procedure. We must also have knowledge of motor nerves in order to provide muscular relaxation and a motionless surgical field and also postoperative persistent neurological defect can be determined by this wisdom.

1. The nerves to scalene and longus colli (C5, 6, 7, 8)

They arise from lower cervical ventral rami almost immediately after emerging from the intervertebral foramina after receiving the respective sympathetic nerve contributions. They supply – Longus colli muscle (C2 - C7) Anterior scalene muscle (C4-C6) Middle scalene muscle (C6-C8) Posterior scalene muscle (C6-C8) Scalenus minimus muscle (C7-C8).

2. Branch to phrenic nerve: C5

A Branch from the fifth cervical nerve joins the phrenic nerve, anterior to the scalenus anterior.

3. The Dorsal scapular nerve: C5

Arises from fifth cervical ventral ramus, pierces scalenus medius, passes behind the levator scapulae and runs to rhomboids. It supplies - Levator scapulae muscle C3-C5 - Rhomboid minor muscle C5 Rhomboid major muscle C5

4. Long thoracic nerve (C5, 6, (7)):

It arises from C5, C6 and C7 in 42% of cases, C5 and C6 pierce the scalenus medius, uniting lateral to it and descends dorsal to the brachial plexus and first part of axillary artery. It crosses superior border of serratus anterior and continue downwards to the lower border of serratus anterior, supplying; branches to each of its digitations. It supplies - serratus anterior muscle. Injury to this nerve causes scapular angle to be drawn medially by unopposed action of rhomboids and levator scapulae and tends to project (winging of the scapula) when horizontal arm is used for forward pushing movements. The arm cannot be raised above the horizontal level.

Branches from Trunks:

1. The nerve to subclavius (C5-6):

The nerve arises from C5 and C6 descends anterior to the plexus and third part of subclavian artery and vein to reach subclavius muscle. Accessory phrenic nerve may occasionally be a branch of this. It supplies to subclavius muscle.

2. The suprascapularnerve (C5-6):

The nerve arises from superior aspect of the superior trunk and runs laterally deep to trapezius and omohyoid, enter supraspinous fossa through the suprascapular notch and supplies: Supraspinatus muscle C5 and Infraspinatus muscles C5-6. Occasionally, it also supplies sensory branches to the shoulder joint, the only sensory fibers that arise above the clavicle. When present, it pierces the deltoid muscle and supply the skin of the proximal third of the arm within the territory of the axillary nerve. Because of its position superior to the plexus, this nerve may be stimulated during the subclavian perivascular technique, giving rise to paresthesia to the shoulder, which cannot be relied upon. Because the nerve leaves the plexus and its investment fascia shortly after arising from the superior trunk and paresthesia in this distribution could indicate stimulation of fibers before or after the nerve has left the sheath.

Infraclavicular branches:

They comprise all of the motor and sensory nerves to the upper extremity proper. Apart from some exceptions, there are no branches arising from the divisions of plexus, Rest of the branches are from the three cords.

Branches from the Cords:

A. Lateral Cord:

1) Lateral Pectoral nerve (C5, 6, 7)

It is larger than the medial pectoral nerve which passes superficial to the first part of the axillary artery and vein, pierces the clavipectoral fascia and supplies the pectoralis major. It sends ramus to the medial pectoral nerve, to supply some fibers to the pectoralis minor. It supplies Pectoralis major muscle (C5-T1).

2) The musculocutaneous nerve: (C5, 6, 7)

The musculocutaneous nerve is the major terminal branch of the lateral cord. After giving off the lateral head to median nerve, it leaves the plexus and enters coracobrachialis muscle. It courses through axillary coracobrachialis muscle and descends obliquely and later between triceps and brachialis, sending motor fibers to the two muscles. It pierces the

deep fascia lateral to the tendon of biceps, just below the elbow and continues as the lateral cutaneous nerve of the forearm. It supplies - Coracobrachialis muscle (C6,7)Biceps muscle (C5,6) Brachialis muscle (C5,6) These are powerful flexor muscles of the forearm, paralysis of which causes inability to flex, supinate and abduct the forearm. The arm hangs in medial rotation; forearm is extended and pronated **-"Erb's paralysis".** The lateral cutaneous nerve of the forearm supplies the skin of forearm's anterolateral surface.

3) The median nerve: (C6, 7, 8, T1)

The nerve arises from two roots, the lateral root of median (from lateral cord) and medial root of median (From the medial cord). The two roots straddle the third part of axillary artery before they unite on its ventral surface. It descends along the course of the brachial artery. In the arm it is first lateral to the brachial artery; medial to the cubital fossa where it is posterior to the bicipital aponeurosis and anterior to the brachialis. It enters the forearm between the heads of the pronator teres, crossing lateral to the ulnar artery. It passes behind a tendinous bridge between the two heads of flexor digitorum superficialis (FDS) descending posterior to this muscle and anterior to flexor digitorum profundus. About 5 cm proximal to the flexor retinaculum (FR), it becomes superficial between the tendons of the flexor digitorum superficialis and carpi

radialis. Then it passes deep to flexor retinaculum into the palm to terminate in muscular and cutaneous branches.

Muscular branches:-

Flexor digitorum profundus, Flexor pollicis longus, Pronator quadratus, Pronator teres, Flexor digitorum superficialis, Flexor carpi radialis, Opponens pollicis, Flexor pollicis brevis and Lumbricals.

Palmar cutaneous branches (Sensory) to Skin of palmar aspect of thumb, the lateral two and middle half finger and distal end of the dorsal aspect of the same fingers. It may encroach upon the area usually innervated by radial nerve, also providing sensory innervations of the dorsal surface of the entire thumb and first three fingers as far as metacarpo phalangeal joint and an area supplied by ulnar nerve and may provide sensory innervation of entire ring finger.

Articular branches are to the elbow joint and proximal radio ulnar joint. Some of intercarpal, carpometacarpal and inter metacarpal joints are said to be supplied by the branches of median nerve. Median nerve injury can occur in forearm, proximal to its muscular and interosseous branches, flexion of second phalanges of all digits is lost, and of the terminal phalanges of index and middle fingers. Terminal phalanges of other two fingers may be flexed by the part of flexor

digitorum profundus, supplied by ulnar nerve. Proximal phalanges may be flexed by the interossei. The thumb cannot be opposed or abducted, nor flexed at its interphalangeal joint. Sensation in the area of distribution is lost. Owing to paralysis of intrinsic pollicis muscles and unopposed action of the extensor pollicis longus an **"apelike" hand** exists. Injury in the mid-forearm may cause only weakness in flexion of the index **("pointing index")** finger, as the branch to flexor digitorum superficialis arise above this level. Injuries proximal to flexor retinaculum cause inability to oppose the thumb. Any condition resulting in reduction in the space below the flexor retinaculum causes pressure on the nerve in the carpal tunnel, between flexor retinaculum and the carpal bones, resulting in pain and slight sensory impairment in the digits supplied and sometimes slight wasting of the thenar muscles. This is called **"carpal tunnel syndrome".**

B. Medial Cord:-

1) Medial head of median nerve (C8, T1)

It joins the lateral head from lateral cord to form the median nerve.

2) The medial pectoral nerve (C8, T1)

It passes between the axillary artery and vein, joins the lateral pectoral nerve, forming a loop around the artery and enters the pectoralis

minor muscle to supply it. Some fibers pass inferiorly to end in pectoralis major. It supplies pectoralis minor muscle C8, T1.

3) The medial cutaneous nerve of the arm (C8, T1)

It leaves the axillary sheath high in the axilla, where part of it forms a loop with the intercosto brachial nerve, with which it has a reciprocal relationship with respect to size and distribution. It supplies the medial portion of the upper arm as far distally as the medial epicondyle. Frequently this nerve innervates the lower portion and the intercosto brachial nerve the upper portion.

4) The medial cutaneous nerve of the forearm (C8, T1)

Initially the nerve is between the axillary artery and vein and supplies a ramus piercing the deep fascia to supply the skin over the biceps almost to the elbow. It travels down the arm medial to the brachial artery, dividing into a larger anterior branch and a posterior branch to supply the skin over the entire medial aspect of the forearm as far as the wrist.

5) The ulnar nerve: (C7) C8 T1

It runs distally through the axilla, medial to the axillary artery, till the middle of the forearm, parallel to and between the median and medial cutaneous nerve of the forearm. Then it angles dorsally and laterally to

descend in groove on the medial head of the triceps. Then it passes behind the medial epicondyle of the humerus, covered only by skin and fascia **("Fussy bone")** and passes down the ulnar side of .the forearm to the hand, dividing into superficial and deep terminal branches.

Muscular branches supply (C8, T1)

Flexor carpi ulnaris, Ulnar head of flexor digitorum profundus, Abductor digiti minimi, Flexor digiti minimi brevis, Abductor pollicis, Palmar interossei, Dorsal interossei.

Articular branches to elbow, wrist joint, intercarpal, carpometacarpal and intermetacarpal joints.

Cutaneous branches supply the skin of the medial two and half fingers of the hand. The ulnar nerve may be injured in the forearm leading to impaired abduction; when attempt is made to flex the wrist. The hand is abducted by the flexor carpi radialis, owing to paralysis of the dorsal interossei, the fingers cannot be spread or flexed at metacarpo phalangeal joints or extended at the interphalangeal joints, and the arm assumes a "clawed" shape from the active opposing muscles. Flexion of the fourth and fifth digits is weakened and the thumb cannot adduct. Wasting of the hypo thenar muscles will occur. Sensation is lost or impaired on the skin supplied by the nerve.

C. Posterior Cord:

1) The upper sub scapular nerve (C5, 6)

2) The thoraco dorsal nerve (C5-6)

3) The lower subscapular nerve (C5, 6)

4) The axillary (circumflex humeral) nerve (C5-6)

It supplies the deltoid, teres minor and the upper part of the long head of triceps. An articular branch supplies the shoulder joint. The axillary nerve is liable to injury in its course around the surgical neck of humerus causing paralysis of deltoid and anesthesia of the skin over the lower part of the muscle. Effective abduction of the arm is not possible.

5) The radial nerve (C5,6,7,8) **It is the largest branch of the brachial plexus**, is the terminal continuation of the posterior cord. It descends behind the third part of the axillary artery. With the profunda artery, it inclines dorsally between the long and medial head of the triceps then passes obliquely across the back of the humerus in the musculo spiral groove and then reaches the lower anterior side of the forearm where its terminal branches arise. Muscular branches supply the extensor compartment of arm, fore arm and hand.

Sympathetic contribution to brachial plexus:

The segmental preganglionic sympathetic contributions are variable, but generally extend more caudal. The highest contribution is usually T2 with T1 contributing only rarely, while lowest may be as far as T8, T9 or even T10. The post ganglionic contributions are from grey rami communicantes from the sympathetic chain.

Cutaneous nerves of upper limb:

Cutaneous branch of lateral supraclavicular nerve supplies skin over the upper half of the deltoid. Lateral cutaneous nerve of the arm (terminal branch of the axillary nerve) supplies the skin over the lower half of the deltoid, lateral, antero-lateral &postero-lateral aspects of the arm. Twigs from the lateral& posterior cutaneous nerves of forearm supply the arm near the cubital fossa at their corresponding areas.

Intercosto brachial nerve supplies a small area near the medial aspect of the axilla. Medial cutaneous nerve of the arm supplies the rest of the medial, antero-medial and posteromedial aspects of the arm .Twigs from the medial cutaneous nerve of the forearm supply the arm near the cubital fossa. Posteriorly a strip of skin along the middle 1/3 is innervated by the posterior cutaneous nerve of the arm. The skin of the front of the forearm is supplied by the medial and lateral cutaneous

nerves of the forearm.

The area of supply of these nerves extends on the back of the forearm. But unlike the arm, major part of the skin of the back of the forearm is supplied by the posterior cutaneous nerve of the forearm**.** (Fig.5)

Cutaneous Nerve Supply of the Hand:

Small part of the palmar aspect of the hand near the wrist is supplied by the lateral and medial cutaneous nerves of the forearm; dorsal aspect is by the posterior cutaneous nerve of the forearm. Major part is supplied by the cutaneous branches of the median, ulnar and radial nerves. The ulnar nerve supplies the palmar & dorsal aspect of medial one and half fingers. The median nerve supplies the palmar aspect of lateral three and half fingers and distal phalanges of dorsal aspect. The rest of the dorsal aspect of lateral 31/2 fingers is supplied by the radial nerve.

Anatomy of the peripheral nerve

Each peripheral nerve axon possesses its own cell membrane, the axolemma, within which is contained the axoplasm. Non myelinated nerves such as autonomic postganglionic fibers are also encased in a Schwann cell sheath. Most motor and sensory fibers are also wrapped in several layers of myelin, a lipoid insulating membrane that separates the axon itself from the Schwann cell sheath. Myelin greatly increases the speed of nerve conduction by producing saltatory conduction via the nodes of Ranvier, which are periodic interruptions in the myelin sheath.

A typical peripheral nerve consists of several groups of axons. Each axon has its own connective tissue covering called endometrium. A group of 100 to 1000 axons are bound together in bundles called fasciculus. Each fasciculus consists of 5-15 layers of fibro elastic tissue called perineurium. This perineurium is thicker at peripheral sites (i.e., at the wrist than at the axilla). The innermost layer of the perineurium is a smooth mesothelial membrane called perilemma. This is the main diffusional barrier. A collection of 5 to 20 fasciculi are surrounded by the outer sheath called the epineurium. This epineurium lies in a matrix called epineural space which consists of loose areolar tissue, nutrient blood vessels, lymphatics and fat.

CLASSIFICATION OF PERIPHERAL NERVES: [7]

Type of fiber	Subclass	Myelin	Diameter µm	Conduction velocity m/s	Location	Function
A	α	+	13-22	70-120	Afferent to& Efferent from	Motor, muscle proprioceptors
	β	+	8-13	40-70	Afferent to& Efferent from muscles & joints	Touch, kinesthesia
	γ	+	4-8	15-40	Efferent to muscle spindles	Touch, excitation of muscle spindles, pressure, muscle tone.
	δ	+	1-4	5-15	Afferent sensory nerves	Pain, temperature-cold, pressure
B		+	1-3	3-14	Preganglionic sympathetic	Various autonomic functions
C	s	-	Less than 2 micron	Less than 2	Post ganglionic sympathetic	Various autonomic functions
	τ-zeta	-	Less than 2 micron	Less than 2	Afferent sensory nerves	Pain, temperature-warm, touch

Structure of the axonal membrane: [7]

Daniell-Davson- Robertson theory says that biologic membranes consist of a bimolecular lipid layers, sandwiched between non lipid proteinaceous monolayers. Probably the most widely accepted current membrane model is that of **Singer and Nicholson**. According to this theory each axonal membrane consists of

1. Lipid matrix (bimolecular in nature) containing long chain fatty acids with polar heads (Phospho tidyl choline and phosphotidyl inositol)

2. Long lipid chain, being oriented towards the middle of the bi lipid layer.

3 Bimolecular lipid layer. This layer contains proteins adsorbed on the surfaces as well as embedded in or spanning the hydrocarbon core. The bilayer character is due to amphophilic phospholipids which have long hydrophobic fatty acyl tails which lie in the center of the membrane, and polar hydrophilic head groups composed of Zwitter ionic (Containing positive and negative charges) components, which project in to the cytoplasm or the interstitial fluid. Within the membrane there is lateral and rotational diffusion, which allows lipids and proteins to migrate in a fluid mosaic. Despite this general behavior, some proteins are fixed with in specific regions of a membrane; sodium channels, for example, are

highly localized at the nodes of Ranvier of Myelinated axons. A dynamic interaction exists between the cell's membrane and cytoplasm. Cytoplasmic enzymes may be regulated by hormone and neurotransmitter receptors in the membrane. The membrane lipids behave as second messengers, and the membrane proteins, are chemically altered and physiologically modulated by intracellular enzymes such as kinases which specifically phosphorylate certain proteins.

PATHWAYS OF PAIN:-

1) From skin and superficial structures:

a. **The laminiscal system:**

The sensation passes from sensory nerve to the dorsal horn of the spinal cord then to lateral spino thalamic tract of other side to the lateral nucleus of thalamus and reaches the post central gyrus of cortex

b. The extra laminiscal system:

Part of the spino thalamic tract goes to reticular formation which is distributed to cerebral hemispheres through the thalamo- cortical projection.

2) From muscles, tendon and bone:

Same pathway.

3) Visceral Pain:

The visceral pain is transmitted through the sympathetic chain and white rami communicantes to the dorsal root ganglia.

4) There is evidence that different types of fibers are carried by the sympathetic nervous system which may not correspond to somatic segment. This may explain the pathway of tourniquet pain. (Fig.6)

Physiology of nerve conduction:

The neural membrane maintains a trans membrane potential of 60 to 90 mV by means of sodium impermeability& potassium permeability. The **'sodium -potassium pump'** (active& energy dependent mechanism) maintains this potential difference by constant expulsion of sodium from the cell. Although the membrane is freely permeable to potassium ions, an intra to extra cellular potassium ratio of 150 mEq to 5 mEq per liter or 30:1 is maintained because of the active exchange of intracellular sodium for extracellular potassium.

According to the Nernst equation, the nerve at rest behaves like a potassium electrode. During normal excitation (depolarization) of a nerve the Na+ permeability increased followed by RMP (resting membrane potential) altered from -90 to -50 mV. This -50 mV is called CTP (critical threshold potential).

At CTP of -50 mV, Na+ influx occurs rapidly with a counter balance of K+ efflux. Because of this RMP reverses from -50 mV to +30 mV at a duration of 0.1- 0.2 m Sec which causes conduction of the depolarization along the entire length of the nerve. At 0.4- 0.6mSec Na+ permeability rapidly decreases resulting Na+ efflux &K+ influx. Therefore RMP of -90 mV reestablished. The ionic fluxes during depolarization and repolarization are passive phenomena, because each ion is moving down its own concentration gradient. The conduction of the impulses is **all or none phenomenon** (if the CTP is not achieved during a local depolarization then conduction of the impulse will not occur.)[25]

LEVOBUPIVACAINE HCL

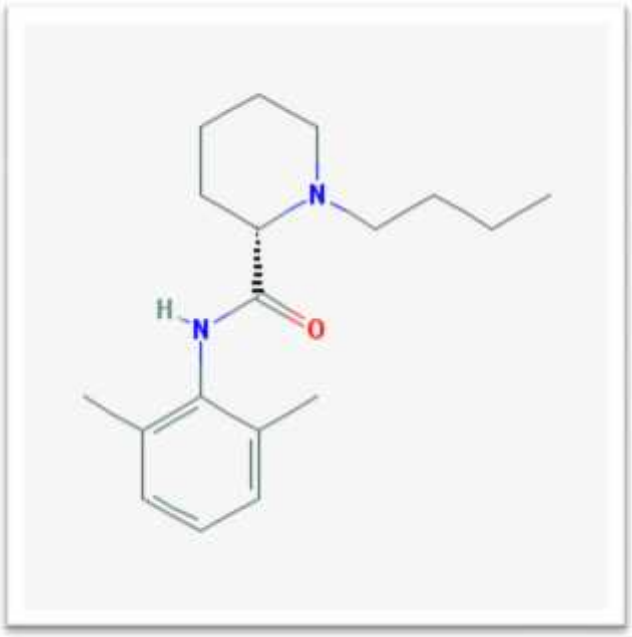


Fig.7

Levobupivacaine is an amino-amide local anaesthetic drug belonging to the family of n-alkyl substituted pipe coloxyl idide. It is the S-enantiomer of bupivacaine, first synthesized by Brian Albert Gennery in the year of 1997 and patented at 2002.

Other Names:

Also known as: Chirocaine, L (-)-Bupivacaine, (S)-bupivacaine, Levobupivacaine, and Novabupi.

CHEMCIAL NAME:

(S)-1-butyl-N-(2, 6-dimethylphenyl) piperidine-2-carboxamide

Molecular Formula: C18 H28 N2 O

PHYSICO CHEMICAL PROPERTIES:

PKa	8.1
Onset	Slow
Relative lipid Solubility	1000
Potency	High
Protein binding	95%
Duration of action	Long.
BNF (British National Formulary) – Max. Dose	150 mg

Levobupivacaine is a colorless solution with a bitter taste and molecular weight288.43 g/mol. It is soluble in water and alcohol and insoluble in ether. It is stable in the presence of alkalis and acids and can withstand heat sterilization and autoclaving.

Advantages of levobupivacaine than racemic bupivacaine include[15, 54]

1. Rapid absorption from the site of injection[32]
2. Rapid onset of action
3. Wide and rapid spread
4. Lesser cardio toxicity[31, 46, 55, 56]

5.Nonirritating, without accumulation of the drug or its metabolic products. [37]

6. Stability permitting heat sterilization.

7. Less vasodilation and has a longer duration of action. It is approximately 13 per cent less potent (by molarity) than racemic bupivacaine.[34]

Target:

Inhibition of sodium channel protein type 10 subunit alpha in the neuronal membrane (Whereas racemic bupivacaine inhibits beta subunits). [23,24]

Action:

Reversible inhibitor of sodium channel.

General Function:

Involved in ion channel activity

Indications

Levobupivacaine is indicated for local anesthesia including infiltration nerve block, labor analgesia[47], ophthalmic, epidural and intra thecal anesthesia in adults and infiltration analgesia in children.

Contraindications

Levobupivacaine is contraindicated for IV regional anesthesia (IVRA).

Adverse effects

Adverse drug reactions (ADRs) [49, 50] are rare when it is administered correctly. Most ADRs relate to administration technique (resulting in systemic exposure) or pharmacological effects of anesthesia (PH, protein binding and relative lipid solubility), however allergic reactions can rarely occur. Systemic exposure to excessive quantities of bupivacaine mainly result in central nervous system (CNS) and cardiovascular effects – CNS effects usually occur at lower blood plasma concentrations and additional cardiovascular effects present at higher concentrations, though cardiovascular collapse may also occur with low concentrations. **CNS effects** may include CNS excitation (nervousness, tingling around the mouth, tinnitus, tremor, dizziness, blurred vision, seizures[16, 38)] followed by depression (drowsiness, loss of consciousness, respiratory depression and apnea).[33, 42] **Cardiovascular effects** include hypotension, bradycardia, arrhythmias and/or cardiac arrest– some of which may be due to hypoxemia secondary to respiratory depression but of less pronounced than racemic bupivacaine.[40, 41, 43]

Peripheral nerve blocks

It was found that giving high concentrations of levobupivacaine (0.5% -0.75%) increases the quality and duration of the block as well as significantly reduces the time of onset. Higher concentrations of levobupivacaine i.e., 0.5%–0.75% speed up the onset, and increase the duration and quality of peripheral nerves blockade (Cox et al) [64]

Like epidural catheter, in nerve blocks also, using continuous catheters and administration of levobupivacaine gave very good results and decreased the systemic opioid requirements in post-op analgesia. Adjuvants like opioids, epinephrine, clonidine and dexmeditomidine, when added with levobupivacaine, will enhance the quality and safety of the block. However since the duration of action of levobupivacaine is longer (10-12 hours), the need of epinephrine has reduced clinical importance. But since epinephrine causes vasoconstriction; it may help in two ways.

1. Reduces the risk of systemic toxicity if there is an accidental overdose.

2. It also signals the inadvertent intravascular injection.[52]

Structural activity relations and physiochemical properties [1, 2]

1) Lipophilic versus hydrophilic balance:

It depends on altering the site of alkyl substitution in the vicinity of either the tertiary amine or the aromatic ring system. Substances of more lipophilic nature are obtained by increasing the size of alkyl substitution. These agents are more potent and longer lasting than their more hydrophilic congeners.

2) Hydrogen ion concentrations:

This depends on the pH, pK-a of the local anaesthetic and pH of the surrounding tissue as mentioned earlier.

3) Minimum anaesthetic concentrations (Cm):

The minimum anaesthetic concentration of the anaesthetic agent necessary to block the impulse conduction along a given nerve fiber within a reasonable standard period of time is termed minimum anaesthetic concentration. The concentration lower than the minimum anaesthetic concentration will not inhibit conduction at all. Minimum anaesthetic concentration values reflect the relative potencies of various drugs. Changes in the electrolyte concentration and acid base status may affect the minimum anaesthetic concentration. Therefore minimum anaesthetic concentration is a standard of local anaesthetic potency,

analogous to minimum alveolar concentration of the inhaled anaesthetic agents. A variety of factors may affect minimum anaesthetic concentration:

a) Fiber size:

Larger the nerve fiber, higher the concentration of local anaesthetic required for inhibition of impulse conduction and their minimum anaesthetic concentration values will be relatively high.

b) PH:

Minimum anaesthetic concentration of a given local anaesthetic is less at a high pH than at a low pH. In a sheathed nerve, Cm for lidocaine is 100 times more/less at pH 7 than at pH 5.

c) Calcium ion concentration:

Local anaesthetic potency correlates directly with the inhibition of calcium binding by phospholipids. The local anaesthetic effect of most of the drugs is inversely proportional to the calcium concentrations.

d) Nerve stimulation rate:

In vitro, individual anaesthetic potency is directly proportional to the rate of nerve stimulation, a greater apparent potency being noted at high stimulation rate.

The electrophysiological effect of local anaesthetics:

The resting membrane potential of a nerve is affected by the various concentrations of the local anaesthetics. As the concentration of the drug applied to the nerve is increased, a decrease in the rate and degree of depolarization is produced. Inhibition of depolarization increases with the time as the concentration of the drug is maintained. Since both the rate of repolarization and the conduction velocity are decreased and the refractory period is prolonged, the number of action potentials that a nerve is capable of transmitting per unit of time decreases as the drug concentration increases until complete block is achieved when the nerve is unable to depolarize to the threshold potential. The more important of the above mechanism is reduction in the rate of depolarization during action – potential development.

The above effects of local anesthetics are a direct consequence of inhibition of membrane conductance of sodium and potassium ions.

Pharmacological effects of local anesthetics:

a) Local -

Cause local nerve blockade and direct effect on smooth muscles.

b) Regional

Loss of pain, temperature, sensation, touch, motor power, vasomotor tone in the region supplied by the nerves blocked.

c) Cardiovascular

Lidocaine and procaine have a stabilizing effect on the cardiac cell membrane. They tend to decrease the automaticity in abnormal or damaged heart fibers and thereby suppress cardiac dysrhythmias. Procaine and procainamide also have quinidine like action, prolong the effective refractory period, slowing conduction and desynchronizing ventricular contraction with consequent depression effect on myocardial contractility. The mechanism involves effect on ionic conduction in myocardium and smooth muscular conducting membrane and on myocardial conducting system.[53]

Over dosage has been associated with ventricular tachycardia, fibrillation and cardiac arrest. There is evidence however that cardiac toxicity does not occur in sub convulsive doses or in the absence of severe electrolyte imbalance or respiratory metabolic acidosis. On vascular smooth muscles, local anesthetics may have local, regional or systemic effect. Usually they cause vasoconstriction at lower concentrations and vasodilatation at higher concentrations. These

changes were associated with simulation and inhibition respectively of tissue bound Ca 2+ ion release. The regional effect is simple vasodilatation in the area supplied by blocked sympathetic nerves. Systemic effects produced by large doses may produce circulatory collapse as a result of medullary depression and of convulsions causing respiratory impairment, rather than because of any direct effect on the circulation.

d) Central nervous system-

Cocaine has undoubted stimulant effects which are unrelated to its local anaesthetic action. The synthetic agents produce sedation and light headedness. Sometimes anxiety and restlessness may occur in which case inhibitory neurons have proved more susceptible than excitatory ones to depression. With more marked toxicity, tinnitus, visual and auditory disturbances, restlessness, slurred speech, nystagmus, shivering muscle tremors.

Progression of dosage and serum level beyond this results in the development of EEG seizure activity with tonic and clonic convulsions followed by CNS depression.[51] Toxic levels probably initially lead to depression of cortical inhibitory pathways, allowing unopposed activity of an excitatory nature. This transitional state of

unbalanced excitatory and inhibitory activity is followed by generalized CNS depression if higher serum levels are reached.

e) Autonomic nervous system

Cocaine has a sympathetic potentiating effect by inhibiting catecholamine uptake. Other local anesthetics do not block nor-adrenaline uptake. Experimentally local anesthetics possess a weak binding effect on cholinergic and adrenergic receptors. The former may account for bronchodilator effect.

g) Neuromuscular junction

They block motor nerves if present in sufficient concentration and the presynaptic blocking effect has been observed under experimental conditions with high procaine, again reflects a membrane stabilizing action.

DEXMEDITOMIDINE

Dexmeditomidine is the d-enantiomer of medetomidine, a substance that has been used for sedation and analgesia in veterinary medicine for many years. It was introduced in 1999 and used as a short-term sedative of less than one day for mechanically ventilated critical care patients. Nowadays it is used as sedative and adjuvant analgesic in the OT, sedation in diagnostic and procedure units, and for other applications such as withdrawal/detoxification amelioration in adult and pediatric patients.

Physicochemical Characteristics[58]

It has a highly selective specificity for shows α2 receptor (α2/α1 1600: 1) whereas clonidine has a ratio of 200: 1 only and therefore it is an absolute α2 agonist. It belongs to the imidazole subclass of α2 receptor agonists and a water soluble drug.

Chemical structure of Dexmeditomedine

Dexmeditomedine hydrochloride is the S-enantiomer of medetomidine and is chemically described as (+)4-(S)-[1-(2, 3-dimethylphenyl) ethyl) -1H-imidazole mono hydrochloride. Has a molecular weight of 236.7 and the empirical formula is C13H16N2 • HCl and the structural formula is:

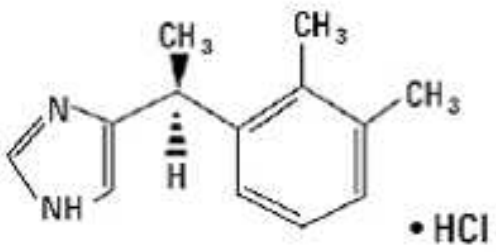


Fig.8

Metabolism and Pharmacokinetics

Dexmeditomidine is rapidly distributed and extensively metabolized in the liver and excreted in urine and feces. It undergoes conjugation (41%), n-methylation (21%), or hydroxylation followed by conjugation. It has 94% protein binding capacity and the concentration ratio of whole blood to plasma is 0.66. Dexmeditomidine has profound effects on cardiovascular variables and may alter its own pharmacokinetics. With large doses, there is marked vasoconstriction, which probably reduces the drug's volumes of distribution. The elimination half-life of Dexmeditomidine is 2 to 3 hours, with a context-sensitive half-time ranging from 4 minutes after a 10-minute infusion to 250 minutes after an 8-hour infusion.

Pharmacology[12, 13]

Dexmeditomidine is a nonselective α2 agonist.α2 adrenoreceptors are membrane-spanning G proteins. Intracellular pathways include inhibition of adenylate cyclase and modulation of ion channels There are three subtypes of α2 adrenoreceptors namely α2A, α2B and α2C. The α2A receptors are found in peripheral blood vessels whereas α2B and α2C are found in the brain and spinal cord. Postsynaptic α2 adrenoreceptors in peripheral blood vessels will produce vasoconstriction, whereas presynaptic α2 adrenoreceptors inhibit the release of norepinephrine. The main response to α2 agonists is because of stimulation of α2B and α2C which are located in the CNS and spinal cord.[19] These receptors are involved in the sympatholysis, sedation, and anti-nociception effects of α2 adrenoreceptors.

Atipamezole:

This is a **s**pecificα2 antagonist. Dexmeditomedine effects are reversed at 75-150µg /kg dose (Sympatho activation). This not approved by US FDA at present because altepamezole is still under trial stages only.

Effects on the Central Nervous System

They are as follows:

1. Sedation

2. Anxiolysis

3. Hypnosis

4. Analgesia

5. Sympatholysis.

Sedation

Sedation is caused due to α2 agonism in the locus caeruleus[10, 11] whereas analgesic action is due to agonistic action on the α2 receptors within the locus caeruleus and within the spinal cord. The quality of sedation produced by Dexmeditomedine seems different compared with that produced by other sedatives acting through the GABA systems. Patients receiving dexmeditomedine infusions as part of their sedation regimen in the postoperative ICU setting have been described as being very easy to wake up and having the ability to follow commands and cooperate while being tracheal intubated. Undisturbed, patients were noted to fall asleep right away. Even though there is marked sedation, the

risk of respiratory depression is very minimal and hence the drug has high safety margins.

Analgesia

The main site of action is the spinal cord.[9, 11]

Cardiovascular effects: [59]

It decreases the heart rate, systemic vascular resistance, myocardial contractility, stroke volume, and blood pressure. By developing highly selective α agonists, it has been hoped to decrease some of these adverse cardiovascular effects and to maximize the desirable hypnotic-analgesic properties. Dexmeditomedine shows a characteristic biphasic response in hemodynamics. An acute IV injection of 2 µg/kg resulted in an initial increase in blood pressure (22%) and decrease in heart rate (27%) from baseline that occurred at 5 minutes after injection. This initial increase in blood pressure is probably due to the vaso constrictive effects of Dexmeditomedine when stimulating peripheral α2 receptors.[14] Heart rate returned to baseline by 15 minutes, and blood pressure gradually declined to approximately 15% below baseline by 1 hour. After an IM injection of the same dose, the initial increase in blood pressure was not seen, and heart rate and blood pressure remained within 10% of baseline.

Infusion of Dexmeditomedine in volunteers also has been shown to result in a compensated reduction in systemic sympathetic tone without changes in baro reflex sensitivity. It also blunts the heart rate and systemic sympathetic activation owing to sweating, but is less effective in blunting cardiac sympathetic response to shivering. Hypotension and bradycardia occurs mainly due to the loading dose. Giving less than 0.4 µg/kg of loading dose and giving slowly over 20 minutes will reduce the severe fall in blood pressure. A frequently reported side effect of Dexmeditomedine has been a dry mouth. Dry mouth is due to a decrease in saliva production.

Uses

1. Short-term sedative for adult intubated patients in the ICU.

2. Because of its proven beneficial effects like anxiolysis, sedation, analgesia, and sympatholysis with minimal respiratory depression, it is very useful in peripheral nerve blocks.[17, 18,20,21,22]

ULTRASOUND

Ultrasound is revolutionizing regional anesthesia techniques that provide anesthesia and postoperative analgesia for patients undergoing surgery. Almost every nerve block previously performed using traditional localization techniques (paresthesias or nerve stimulation) can now be performed using real-time ultrasound guidance. Unlike other localization techniques, ultrasound allows the visualization of nerves and surrounding structures as well as visualization of the needle and local anesthetic.

Basic Ultrasound Physics:

Ultrasound refers to high-frequency waves produced by passing electricity through piezoelectric elements. These elements vibrate at a high frequency creating ultrasound waves. It is therefore mechanical vibrations of matter-sinusoidal waves-propagated at frequencies inaudible to the human ear (> 20,000 Hz). The human ear hears sounds between 20 Hz (low sound) and 20,000 Hz (High pitched sound). We talk about ultrasound when the vibration frequency is greater than 20,000 Hertz (Hz), and generally ultrasound used in medicine has frequencies of above 1 MHz (between 1 to 25 MHz). The waves leave the ultrasound probe and enter the body. These waves can then be reflected, refracted, scattered, or absorbed depending on what internal structures they encounter. (Fig.9)

The ultrasound probe senses the reflected ultrasound waves, and ultrasound images are generated from these reflected waves.

If a needle or nerve is 90 degrees perpendicular to the ultra- sound wave, it will appear much brighter on the ultrasound image than a needle or nerve at 45 degrees to the ultrasound wave.

Probe Selection:

The type of sensor that is used determines the quality of the resulting image and the depth at which one can visualize anatomical structures.

The high frequency linear probes (10- 18 MHz) are used to perform superficial blocks with high resolution images. Penetration does not exceed a 6 cm depth.

The low frequency convex probe of 3-8 MHz allows the construction of deeper blocks, penetrating 10 -20 cm depth. It also allows a greater field of vision with a small contact area due to the layout of the surrounding crystals. (Fig.10)

Frequency:

Most sensors can emit at different frequencies. This frequency should be adjusted on a case by case basis, depending on the depth of the block to achieve and on the echogenicity of the patient. In general for a very superficial block, it is often useful to choose a very high frequency and the opposite for a deep block.

Machine Settings:

There are only three settings to remember

- General (Gen): General imaging frequency—this is best for most blocks.
- Resolution (Res): High-frequency imaging—this is best for shallow blocks.
- Penetration (Pen): Low-frequency imaging—this is best for deep blocks.

For each probe, the image quality and penetration can simply be adjusted by using the Gen/Res/Pen settings (Fig.11).

Depth:

The depth should be adjusted so the nerve target is in the middle of the screen. Most ultra- sounds are preset with the focal zones in the middle of the screen. Focus allows the best lateral resolution possible, improving image quality. This means you will have the clearest image of your target if it is in the middle of the screen. Use the depth settings so the needle and nerve are in the middle of the screen when possible. Some machines require manual adjustment of focal zones.

Focus:

Ultrasound beams can be focused much like light can be focused through a lens on a camera. Just like a photograph, when ultrasound

images are out of focus, images can appear less sharp. Correct focus improves lateral resolution.

Focus position affects image quality. The identical inter scalene anatomy is scanned on 2 images. On the left the focus is set deep and on the right the focus is set shallow. The nerves are easier to identify on the right, where the focus is set at the same depth as the nerve roots in the inter scalene groove (Fig.12).

Gain:

Gain adjusts screen brightness. There are no specific rules to adjust the gain. Usually, each person will have a preference for gain settings. Some general suggestions include the following:

1. The brightness of the screen should be adjusted so vascular structures appear dark or anechoic.
2. Too much gain results in artifacts such as reverberation that can "repeat" bright structures, such as fascial planes and obscure targets.
3. Because ultrasound beams returning from deeper structures become attenuated (or return a weaker signal), increasing the distal gain can be helpful.

Over- and under-gained images:

The center image demonstrates the bright radial nerve in the center. The under-gained image of the same nerve above is very dark, and the

over-gained image below is very bright. Both over- and under-gain lose vital detail (Fig.13 and 14).

Color Doppler:

Color Doppler allows visualization of flow. Flow can be arterial, venous, or even from injection of local anesthetic.[26] It is very important to remember that the red or blue color on the screen does not signify oxygenated (arterial) blood or deoxygenated (venous) blood. If a structure appears red when using color Doppler, this signifies that the fluid is moving toward the probe. If a structure appears blue when using color Doppler, the fluid is moving away from the probe (Fig.15).

Sometimes there will be no color in a structure that may be a blood vessel. The Doppler principle works best when the probe is parallel to the blood flow, and there must be an angle between the flow of the blood and the probe of less than 90 degrees. The Doppler equation uses the cosine of the angle between the probe and the flow. The cosine of 90 is 0. This means if the probe is at 90 degrees to the calculated flow, the measured blood flow will be zero and there will be no color on the screen. The probe must be tilted in one direction or the other to better visualize blood flow with color Doppler. When performing nerve blocks, it is often beneficial to move the Doppler box not only over the large artery but also over the path the needle will take toward the nerve. Moving the Doppler

box over the projected needle path prior to needle insertion will help identify smaller vessels and prevent accidental vascular puncture.

How to visualize nerves and needles?

Long Axis vs. Short Axis

The term axis in ultrasound guided regional anesthesia is used to describe the view obtained of a structure (nerve or vessel) in relation to the ultrasound beam. A long-axis view is an image along the length of the nerve, and a short-axis view cuts across the diameter of the nerve. In ultra- sound guided regional anesthesia, we generally try to obtain a short-axis view of the nerve.

In-Plane vs. Out-of-Plan

The term “plane” in ultrasound guided regional anesthesia is used to describe the needle position relative to the ultrasound beam. Most nerve blocks are performed with an in-plane approach. If performed correctly, in-plane approaches allow the entire needle (shaft and tip) to be visualized. This allows the user to place the needle tip with the greatest amount of confidence and, potentially, the greatest amount of safety. Out-of-plane needle approaches most closely resemble needle approaches used for many years when paresthesias or nerve stimulation was used to locate nerves. Some people prefer out-of-plane needle approaches because they are more comfortable with the traditional approaches to

locate nerves. However, out-of-plane nerve blocks do not visualize the tip of the needle at all times and, therefore, potentially do not offer the same level of confidence and potential safety that comes with always knowing where the needle tip is located (Fig.16).

Out-of-Plane Technique (sliding)

The probe should be held over the target. Now the needle is advanced slowly in shallow plane with the bevel up (to make it maximally visible). We should look carefully for the hyper echoic dot as the needle cuts the plane of the beam. Advancing of the needle is stopped immediately. The probe is slide forward beyond the needle tip. Needle angle is increased appropriately and now the needle is advanced again, looking carefully for the hyper echoic tip. As soon as the needle tip is visible, we should stop the needle. The process is repeated until the needle descends down on to the target (Fig.17).

Out-of-plane technique (tilting):

The probe should be held over the target with the probe tilted away from the needle. This increases the reflection from the needle and allows room to tilt the probe toward the needle as it is advanced. Now the needle is slowly advanced in a shallow plane with the bevel up. Any hyper echoic dot should be carefully looked out as the needle tip appears on the screen. As soon as the needle is visible, it should be stopped immediately. Now the probe is tilted to ensure that the ultrasound plane is

just beyond the tip of the needle. Next the needle is advanced toward the target. Again, as soon as the needle breaks the plane of the beam, it should be stopped and repeat the process until the target is reached (Fig.18).

Out-of-plane technique

The needle should be adjusted. Probe should be held over the target. The needle is advanced under the middle of the probe with a shallow insertion angle. When the tip of the needle appears visible as a bright dot on the screen, immediately needle advancement should be stopped. Now the needle is withdrawn and redirected at a steeper angle. We should advance until the tip is seen and stop again. The process is repeated at steeper angles until the target is reached. The hyper echoic tip can be viewed in a stepwise fashion as it descends toward the target (Fig.19).

Visualizing the Injection

Probably the best marker for ruling out intravascular injection of local anesthetic is ultrasound visualization of inject ate. If local anesthetic spread is not obvious on the ultrasound screen, injection should be immediately halted and needle tip position re-confirmed. Needle is assumed intravascular if no spread of the local anesthetic is visualized.

Dropout artifact:

This is caused because the entire foot of the probe is not in contact with the skin surface (Fig.20).

Attenuation occurs as sound waves penetrate tissues. On the left the image is dark, and on the right the distal gain has been increased to make the distal portion of the image more visible. Other techniques to view deeper structures are reducing the frequency and adjusting the focus to a deeper position (Fig.21).

Needle misalignment is common and can lead to incorrect identification of the needle tip. In the two ultrasound images the needle has not moved, but because of misalignment the needle tip position appears different (Fig.22).

Bayonet Effect:

The needle appears to bend as it passes through muscle and into the local anesthetic pool (Fig.23).

Reverberation Artifact

When the needle is completely in-plane, it will have a shadow below it. The ultrasound beam is bouncing between the anterior and posterior walls of the needle, creating a “shadow” effect below the needle (Fig.24).

Intra neural Injection:

Injection near a nerve should produce a dark (anechoic) area of local anesthetic around a nerve. Occasionally, we may get too close to a nerve and actually insert the needle inside the nerve. During injection of local anesthetic, the patient may or may not feel a paresthesia. If the nerve does not expand during injection, the needle is likely outside of the nerve. If the nerve expands during injection, the needle is likely intra neural and injection should be immediately stopped and the needle withdrawn from the nerve. Current thinking is that if the injection is sub-epineural (outside covering of a nerve bundle) then there is only a small chance for permanent injury. If the injection goes sub-perineural (i.e., inside a nerve fascicle), then the likelihood of prolonged neuropathy is increased (Fig.25).

Positioning of the patient:

There are several important qualities to consider when positioning a patient for an ultrasound guided nerve block:

1. Ultrasound probe and needle position—In-plane needle approaches usually require slightly longer needles and, therefore, more space is required to advance the needle.

2. Ultrasound machine location—Place the machine between the operator and the patient so the patient, the screen, and the needle can be easily viewed with minimal repositioning.

3. Patient will be sedated.

When starting ultrasound guided regional anesthesia, most operators use the dominant hand to advance the needle and the non-dominant hand to hold the ultrasound probe. His hands should rest comfortably during the block to steady the probe and needle.

Equipment and preparation:

Needle Type: Echogenic needles include

1) A 50 mm extended tube with an insulated needle. The tube that can be easily attached with a syringe of local anesthetic,

2) Skin Preparation-Chlorhexidine, Betadine or alcohol.

3) Probe Cover For continuous indwelling catheters, full drape and full probe covers are recommended to maintain strict aseptic technique. For single-injection nerve blocks, probes can be covered with a clear adhesive dressing (Fig.26).

Monitors:

Standard monitors for nerve block procedures are

a) Pulse oximeter,

b) ECG monitor (three leads) and

c) Noninvasive blood pressure.

Time Out

Prior to block placement, a “time-out” should be performed to check for correct

1. Patient identification

2. Surgery and surgery side

3. Block and block side

4. Monitors and equipment available

5. Patient position.

OBJECTIVES

This is a study conducted to compare the effects of addition of Dexmeditomedine to Levobupivacaine for Axillary brachial plexus block. The effects will be studied in terms of:

- Onset of sensory blockade and motor blockade.
- Duration of sensory and motor blockade
- Complications / side effects if any

REVIEW OF LITERATURE

History of brachial plexus block

The first brachial plexus block was performed by **William Stewart Halsted** in1885, less than a year after **Koller** demonstrated the anaesthetic properties of cocaine on the eye of a patient. Halsted exposed the roots surgically under local Infiltration and injected each of them with a small amount of dilute cocaine (0.1%) intra neutrally under direct vision.(Fig.7) Only about 0.5 ml of local anaesthetic was required to produce complete anesthesia. In 1897, **Crile** used a similar technique in which the plexus was exposed under local anesthesia. Just behind the sterno mastoid muscle and cocaine injected into the nerve trunks under direct vision which has done as a therapeutic measure in a 12 year old boy who developed tetanic spasms following a compound fracture of the forearm, later the technique was used to provide anesthesia for upper arm surgeries.

1. Ishrat Hussain Mir, Abdul Hamid (2008) conducted a study on adding butorphanol with lignocaine in axillary brachial plexus block and demonstrated that adding butorphanol with lignocaine for brachial plexus blockade provides a significant prolongation of the blockade.

2.Kenanet al[63]concluded that adding Dexmeditomedine to Levobupivacaine in axillary brachial plexus block shortens sensory block

onset time, increases the sensory and motor block duration and time to first analgesic use, and decreases total analgesic use with no side effects.

3. In a study by Sarita et al [22], they concluded that Dexmeditomedine prolongs the duration of sensory and motor block duration of analgesia and enhances the quality of block as compared with clonidine when used as an adjuvant to Bupivacaine in peripheral nerve block.

4. Aliye esmaoglu et al (2010) evaluated the effect of adding Dexmeditomidine (100µg) to 0.5% Levobupivacaine for axillary brachial plexus blockade. The primary endpoints were the onset and duration of sensory and motor block and duration of analgesia. They concluded that Dexmeditomidine added to Levobupivacaine for axillary brachial plexus block shortens the onset time and prolongs the duration of the block and the duration of postoperative analgesia.

5. Obayah et al[61] studied the effect of Dexmeditomedine on the duration of sensory blockade. They evaluated the effect of adding 1µg/kg of dexmeditomedine to 0.25% bupivacaine on the duration of postoperative analgesia in children who underwent repair of a cleft palate. Conclusion of their study was Greater palatine nerve block with a combination of Dexmeditomidine and bupivacaine increased the duration of analgesia after repair of a cleft palate by 50% with no clinically relevant side effects.

6. Rachana Gandhi, Alka Shah and Patel conducted a prospective double blind study to compare the postoperative analgesic efficacy and safety of Dexmeditomidine (30µg) for brachial plexus blockade along with bupivacaine (0.25%). Assessment of motor and sensory blockade, pulse, systolic blood pressure, respiration and side effects were noted every 5 minutes for first 30 minute and every 10 minute till end of surgery. Duration of analgesia and incidence of various complications following the procedure were observed. It was observed that in control group onset of motor and sensory blockade was faster. Dexmeditomedine group had better hemodynamic stability and greater postoperative analgesia.

7. In a study of Amany S et al, [21] 0.75 µg/kg of Dexmeditomidine and plain bupivacaine were compared in USG guided infraclavicular block. They found that dexmeditomidine reduced the time of onset of sensory and motor block, increased the duration of analgesia, reduced the VAS pain scores and finally decreased the supplemental needs of opioids.

MATERIALS AND METHODOLOGY

Source of data:

A comparative study of USG guided axillary brachial plexus block with levobupivacaine and levobupivacaine with dexmeditomedine was carried out at Govt. Mohan Kumaramangalam Medical College Hospital (GMKMCH) attached Govt. Mohan Kumaramangalam Medical College, Salem. The patients belonged to the inpatient section of the Departments of Plastic Surgery and Orthopedic Specialty. Sixty patients of the age group 18 to 60 years of both sexes requiring both elective and emergency surgery of the elbow, forearm &hand were selected and divided into two groups of 30 patients each.

Methods of collection of data

Patients undergoing elective forearm and hand surgery will be included in the study, after obtaining the ethical committee clearance.

Inclusion criteria

Patients belonging to age group 18-60 years with ASA grade I and grade II undergoing elective operative procedure for upper limb surgeries (i.e. elbow & forearm and hand surgeries.)

Exclusion criteria

1. Patients who refuse.

2. Patients with history of bleeding disorders.

3. Patients with local infection at the site of block.

4. Patients with documented neuromuscular disorders.

5. Patients with respiratory compromise.

6. Patients with known allergy to local anaesthetic drugs.

7. ASA grade III and IV patients.

Mode of selection of cases:

Randomized prospective double blinded case control study

Allocation to different regimens:

Group A: Patients receiving 0.25% Levobupivacaine (40ml) + normal saline (0.5ml).

Group B: Patients receiving 0.25% Levobupivacaine (40ml+ Dexmeditomidine (0.5ml).

The same volume of saline (placebo) corresponding to that of Dexmeditomedine, was added to Levobupivacaine for the Levobupivacaine + placebo group (Group A).

Sample size:

30 patients are taken as study and 30 patients are taken as control (total 60 patients).

Statistical data analysis:

Detailed descriptive analysis of socio demographic and clinical parameters was done in the first step. The quantitative variables were presented as mean ± standard deviation and the categorical variables were presented as frequency and percentage. All the basic parameters were compared between the two treatment groups. The time taken for onset of anesthesia, duration of anesthesia, analgesic requirement etc. was considered as primary outcome parameters. The hemodynamic and respiratory parameters of the patients during and after anesthesia were considered as secondary outcome variables. Both the outcomes were compared between the two treatment groups, by calculating mean differences. The statistical significance and 95% CI of these differences was assessed by unpaired t-test. Microsoft excel and IBM SPSS (Statistical Package for Social Sciences) version 21 were used for statistical analysis Unpaired t-test was applied for demographic data, hemodynamic parameters, onset and duration of sensory and motor blockade and duration of analgesia. For assessing the quality Fisher exact test was applied. P-value was considered significant if <0.05 and highly significant if <0.001.

Equipment for axillary brachial plexus block:

1. 50mm insulated needle with catheter.

2. 10ml syringe.

3. Local anaesthetic agent – Levobupivacaine without adrenaline /

Levobupivacaine with dexmeditomedine 0.5µg /kg

4. USG machine – Logiq- E by GE Health Care

High frequency small linear probe of 12 MHz

Procedure:

The procedure was explained to the patient and informed consent was obtained. All aseptic precautions were taken throughout the procedure. All the necessary equipment and emergency drugs were kept ready for resuscitation in order to manage the toxic and untoward reactions occurring during the procedure. The patients were brought to the operation theatre and advised to lie in supine position with due comfort on the tilt able (operating) table.

Pre-op heart rates (HR), Noninvasive blood pressure (NIBP), Saturation pressure of oxygen (sPO_2) were recorded. Intravenous access was secured in the non-operative limb and a crystalloid was started. An anesthesiologist experienced in performing ultrasound guided nerve blocks gave the axillary block. He was blinded from the drug composition of the local anaesthetic mixture used for the axillary block.

60 patients were randomly allotted into 2 groups, group A and group B. All the patients received injection Midazolam 0.05mg/kg and injection Fentanyl 0.5µg/kg intravenously15 minutes before the procedure.

Axillary brachial plexus block was given with the patient lying supine with the arm abducted from the body (90°) and flexed in the elbow joint(90°)[8]. The probe of ultrasound machine was placed over the axillary region and axillary sheath will be identified. It was approached using a 50 mm insulated needle with catheter for injecting the LA solution. After repeated negative aspirations, 10 mL of local anaesthetic solution which contains either Levobupivacaine alone or Levobupivacaine with Dexmeditomedine was injected at each nerve (radial, ulnar, median, musculoctaneous). Totally 40 ml given with either 0.5 ml normal saline or 0.5 ml of Dexmeditomedine which is 25 µg. A thorough massage was given so that the drug distribution would be better.

The onset and duration of sensory loss and motor blockade were studied. The loss of pinprick sensation was checked every 3 minutes till the onset of loss of sensation and then every ½ hourly till the regain of sensation. The motor blockade was assessed every 3 minutes till the loss of movements and then every ½ hourly till the regain of movements.

Blood loss was assessed and IV fluids were given according to the loss. Duration of surgery has been recorded. The intra- and post-operative assessment was done by an anesthesiologist who had no idea of the drug given.

Parameters of comparison:

1) **Onset of analgesia:**

This was recorded as the interval between the time of injection and the development of loss of sensation to pin prick. The dermatome areas corresponding to the median nerve, radial nerve, ulnar nerve and

musculo cutaneous nerves were checked at every minute till there was complete loss of sensation.

Grading's of sensory block:

Grade 0: Feeling of Sharp, pin prick sensation.

Grade 1: Analgesia (feeling of dull sensation).

Grade 2: Anesthesia (feeling of no pain at all)

2) Quality of analgesia:

The onset and completion of analgesia was tested by loss of sensation to pin prick. The effect of analgesia after injection was graded as:

Grade I: Good analgesia, sedatives were given only to relieve apprehension.

Grade II: Inadequate, incomplete or patchy analgesia, supplementation was given with N2O/O2, fentanyl, midazolam or ketamine.

Grade III: Very poor analgesia. General anesthesia was administered.

The conclusion of Grade 11 was arrived when any one of the segments supplied by four major nerves (radial, ulnar, median and musculocutaneous nerves) did not have loss of sensation even after 30 minutes of the block.. They were supplemented with mask ventilation with nitrous oxide, IV ketamine 0.5 mg/kg / fentanyl (1µg/ kg) and midazolam (0.02 mg/kg). When there was no loss of sensation in more than one nerve segment then it was considered a failed block. In such case, general anesthesia was provided. Sedation component was recorded by the Ramsay Sedation Score.

2) **Degree of motor blockade:**

The duration of motor block was called as the time interval between the end of giving the drug and the recovery of complete motor function of the elbow, wrist and finger movements.

Modified Bromage scale for upper extremities (3-point scale):

Grade I: Complete block, no active movement of entire elbow, forearm and hand.

Grade II: Almost complete block, slight active movement of the fingers retained.

Grade III: No block, nearly full range of movement retained.

4) Duration of analgesia

Duration of analgesia was recorded with the help of Visual Analog Scale (VAS) which ranges from 0 to 10.This scale was noted per every 60 minutes post-operatively till it comes to 5. Then the rescue analgesia was provided. The drug used was injection diclofenac sodium (1.5 mg/kg) intramuscularly. The time of administration was recorded.

The duration of sensory block was called as the time interval between the end of drug injection and the complete resolution of pin prick sensation on all nerve segments.

5) Complications: All patients were monitored for complications (if any) during the intra-operative period and up to 48 hours post-operatively. The observations and particulars of each patient were recorded in the proforma enclosed.

RESULTS AND OBSERVATION

A comparative study of Levobupivacaine alone and Levobupivacaine with Dexmeditomidine in axillary brachial plexus block was carried out on 60 patients divided into 2 groups of 30 patients each in the age group of 18to60 years. The following observations were made.

Results:

A total of 60 participants were included in the final analysis. Out of the 60, 34 (56.7%) were randomized to intervention group A, to receive 0.25%Levobupivacaine+Normal saline (0.5 ml)). The remaining 26(43.3%) received intervention group B i.e. 0.25% Levobupivacaine + Dexmeditomedine (0.5ml). (Table1)

Table1:

Descriptive Analysis Intervention groups (N=60)

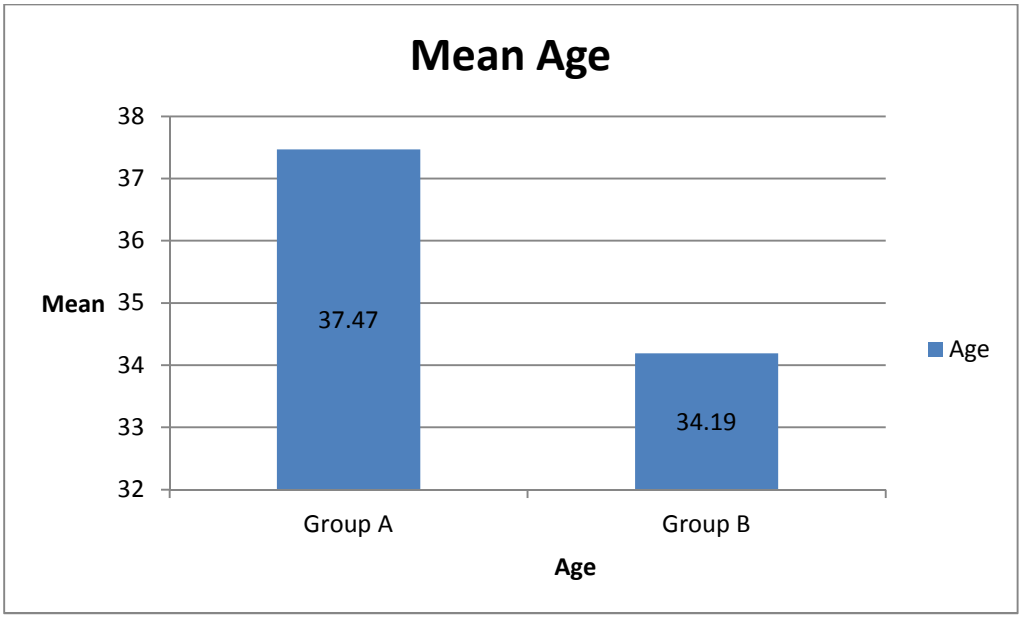
Treatment group	Frequency	Percent
Group A (0.25%Levobupivacaine+Normal saline(0.5 ml))	34	56.7
Group B (0.25%Levobupivacaine+Dexmedetomidine(0.5ml))	26	43.3
Total	60	100

The socio demographic and anthropometric parameters were comparable between two groups. Only minor differences existed in the mean values of age and proportion of females. (Table2)

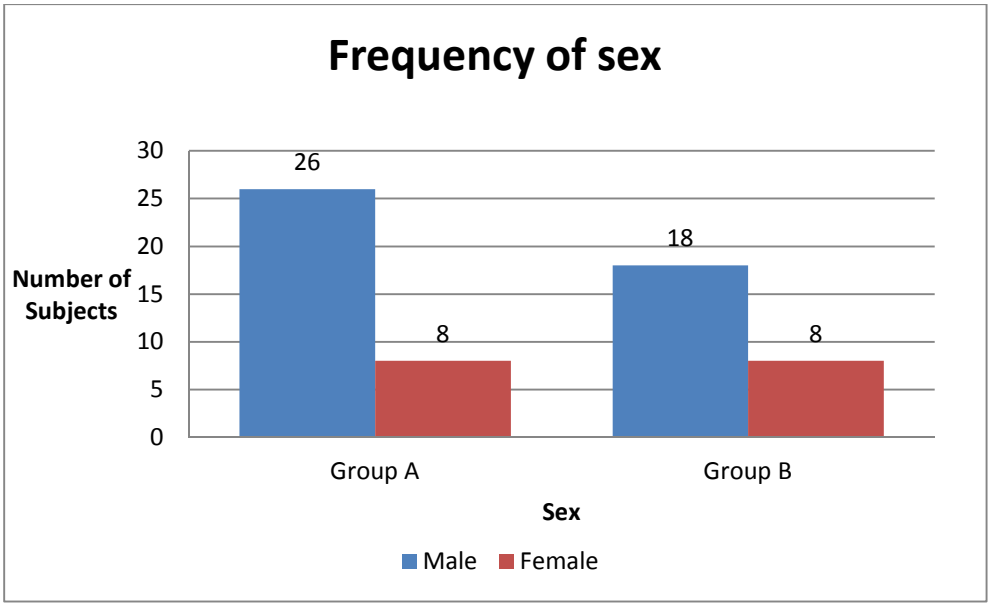
Table2:

Descriptive Analysis of Socio demographic parameters in study groups (N=60)

Parameter	Group A	Group B
Age [mean(SD)]	37.47(SD)	34.19 (SD)
Sex		
Male [Frequency (%)]	26 (%)	18 (%)
Female [Frequency (%)]	8 (%)	8 (%)
Anthropometry		
Weight [mean(SD)]	67.59 (SD)	1.67(SD)
Height [mean(SD)]	67.85 (SD)	1.68(SD)



Comparison of age distribution between two groups.



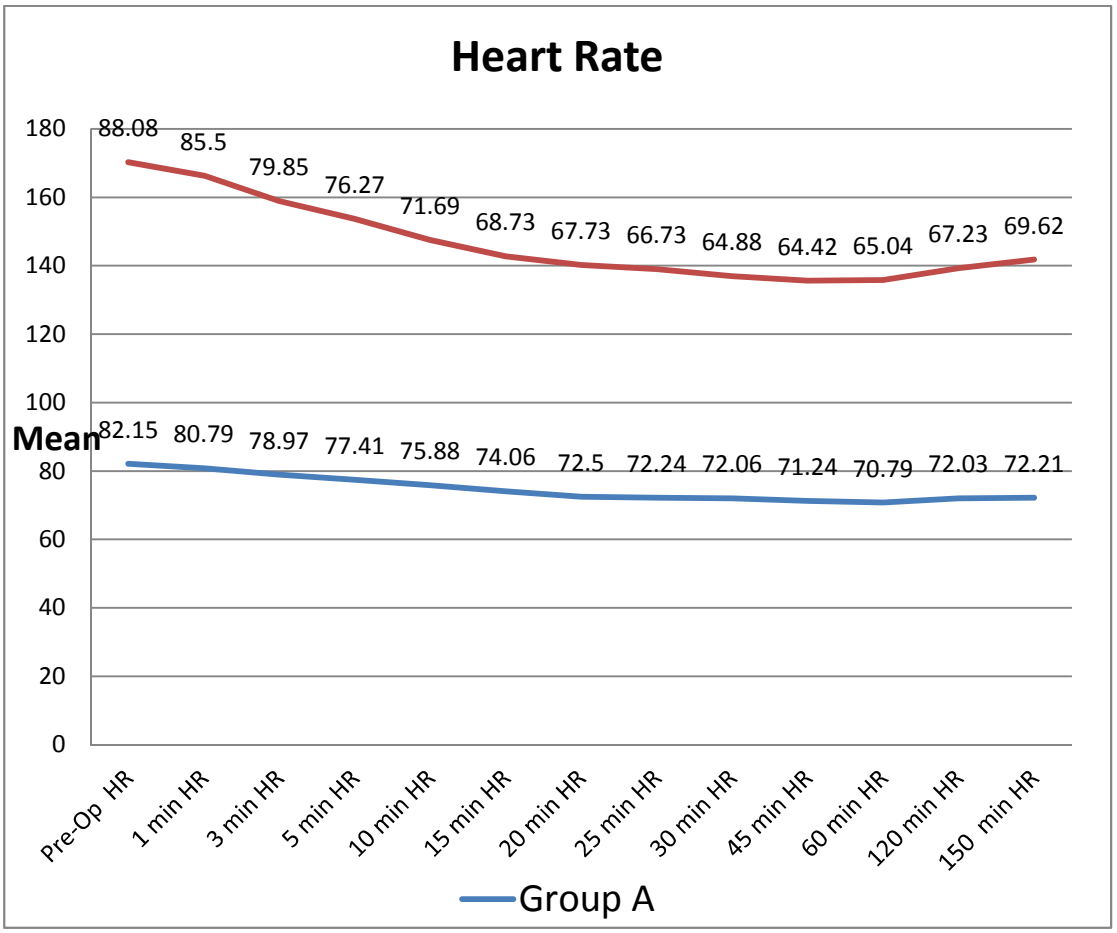
Comparison of sex distribution between two groups.

There was no statistically significance difference in the baseline hemodynamic and respiratory parameters in both the study groups. So the two study groups were comparable in terms of baseline hemodynamic parameters. (Table 3)

Table3:

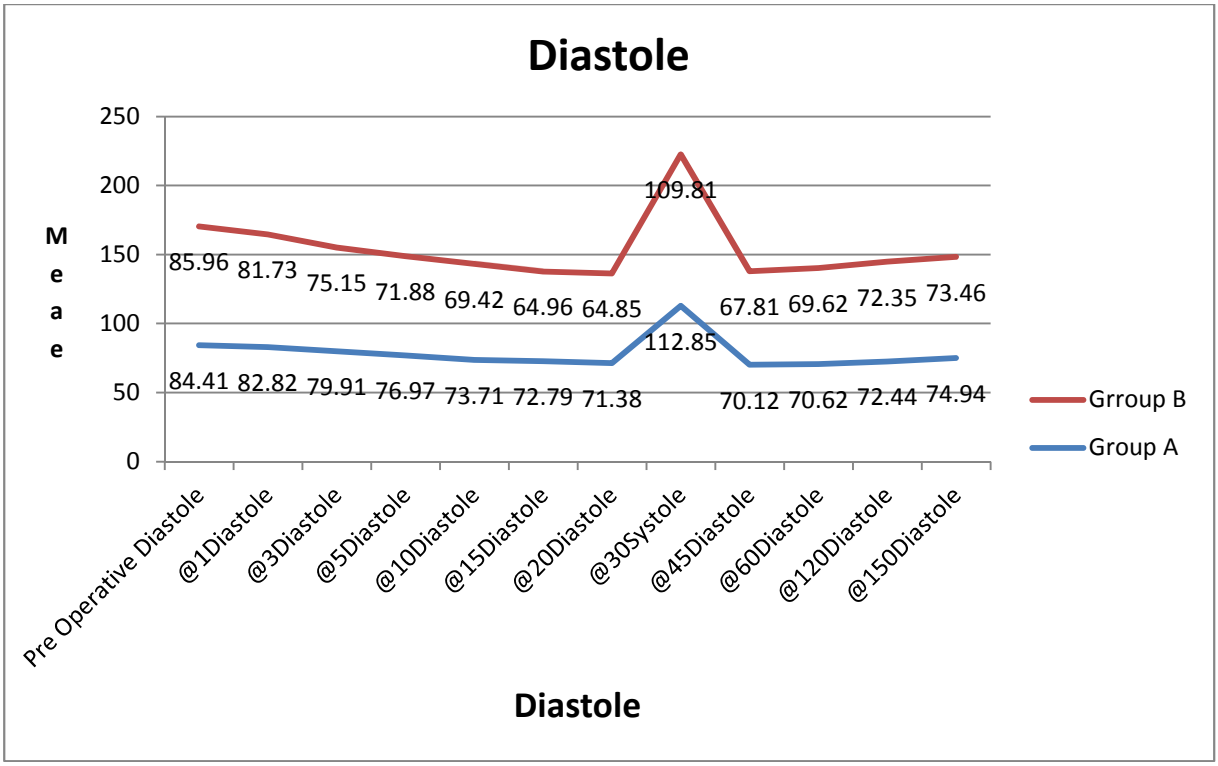
Baseline (pre-operative) hemodynamic and respiratory parameters in both groups (N=60)

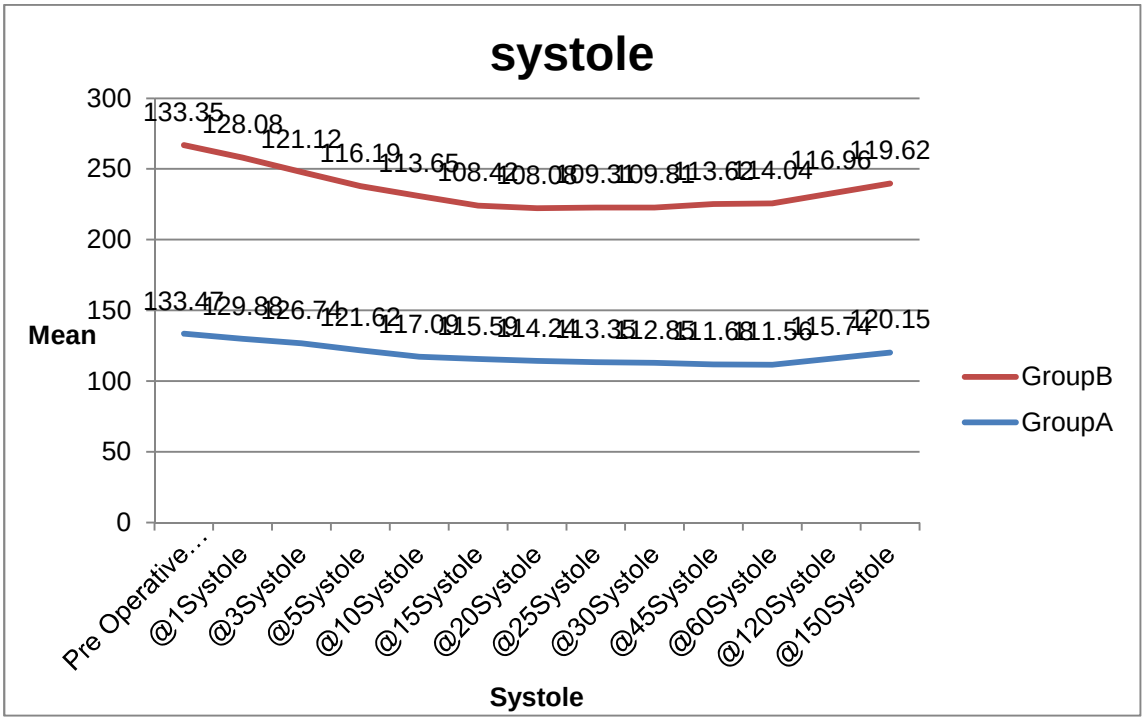
Parameter	Group	Mean	Mean difference	P value	95% CI	
					Lower	Upper
Pre-Operative Heart Rate	Group A	82.15	-5.93	0.123	-13.519	1.659
	Group B	88.08				
Pre-Operative Systole	Group A	133.47	0.124	0.980	-9.563	9.812
	Group B	133.35				
Pre-Operative Diastole	Group A	84.41	-1.550	.566	-6.922	3.823
	Group B	85.96				
Pre-Operative Respiratory rate	Group A	13.47	-.645	.187	-1.611	.321
	Group B	14.12				
Pre-Operative SPO2	Group A	99.71	-.025	.915	-.490	.440
	Group B	99.73				



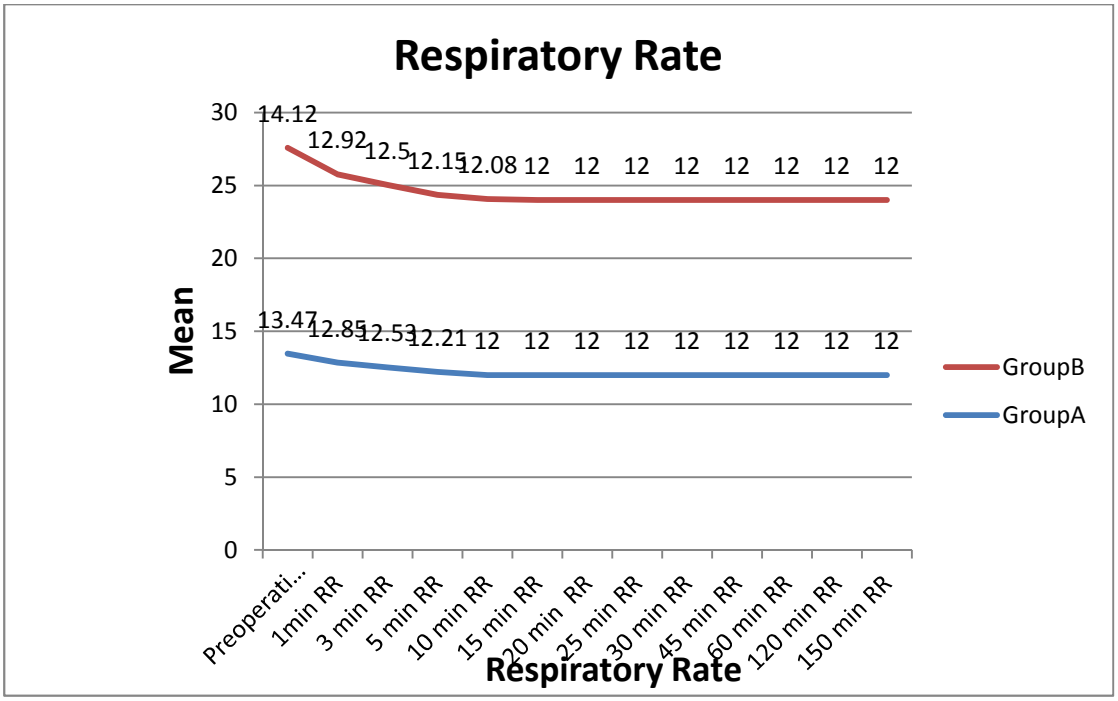
Comparison of pre-op heart rate between two groups

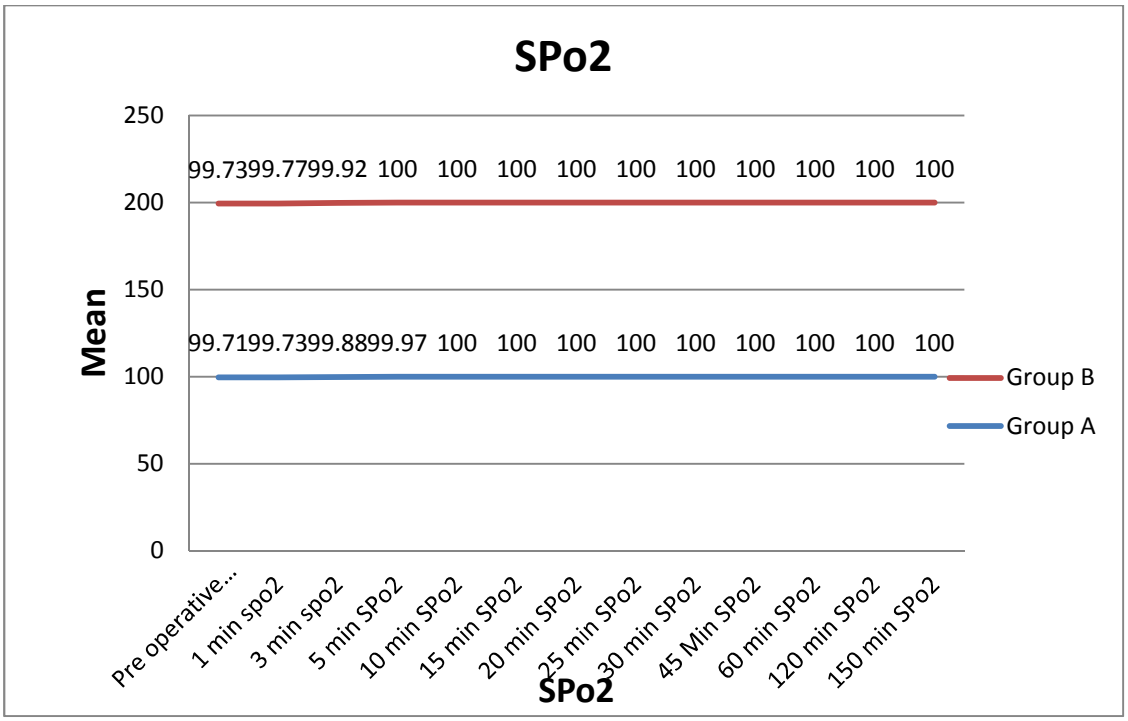
Comparison of pre-op blood pressure between two groups





Comparison of pre-op respiratory parameters between two groups


Respiratory Rate
30
25
20
15
10
5
0
Mean
14.12
12.92
12.5
12.15
12.08
12
12
12
12
12
12
12
12
13.47
12.85
12.53
12.21
12
12
12
12
12
12
12
12
12
Preoperati...
1min RR
3 min RR
5 min RR
10 min RR
15 min RR
20 min RR
25 min RR
30 min RR
45 min RR
60 min RR
120 min RR
150 min RR
Respiratory Rate
GroupB
GroupA


SPo2
250
200
150
100
50
0
Mean
99.73
99.77
99.92
100
100
100
100
100
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100
100
100
100
99.71
99.73
99.88
99.97
100
100
100
100
100
100
100
100
100
Pre operative...
1 min spo2
3 min spo2
5 min SPo2
10 min SPo2
15 min SPo2
20 min SPo2
25 min SPo2
30 min SPo2
45 Min SPo2
60 min SPo2
120 min SPo2
150 min SPo2
SPo2
Group B
Group A

The onset of sensory blockade (mean difference 0.04 minutes, p-value <0.001) and motor blockade (mean difference 0.03 minutes, p-value <0.001) were quicker in group B compared to group A. Both these findings were statistically significant. Both the duration of sensory blockade (mean difference 4.7 hours, p value <0.001), and motor blockade (mean difference 1.8 hours, p value <0.001) were longer in group B compared to group A and both these findings were statistically significant. (Table 7)

Table7:
Comparison of onset and duration of anesthesia in both study groups (N=60)

Parameter	Group	Mean	Mean difference	P value	95% CI	
					Lower	Upper
Onset of Sensory Blockade (minutes)	Group A	0.07	0.04	<0.001	0.032	0:048
	Group B	0.03				
Onset of Motor Blockade	Group A	0.10	0.03	<0.001	0.023	0.037
	Group B	0.07				
Duration of Sensory Block	Group A	8.91	-4.70	<0.001	-5.31	-4.09
	Group B	13.61				
Duration of Motor Block	Group A	5.82	-1.82	<0.001	-2.37	-1.28
	Group B	7.65				

Time taken for starting of regression (mean difference -1.37 minutes, p-value <0.108) was more in group B compared to group A, but this finding was not statistically different. All other parameters related to duration of anesthesia including time taken for full motor and sensory recovery were longer in group B compared to group A. These differences were statistically significant. (Table 8)

Table8:

Comparison of other anesthesia related parameters in both study groups (N=60)

Parameter	Group	Mean	Mean difference	P value	95% CI	
					Lower	Upper
Time Taken for starting of Regression	Group A	0.53	-1:37	.108	-3:37	0:22
	Group B	2.31				
Time of full sensory recovery (mnts)	Group A	10.01	-4.78391	<0.001	-5.42451	-4.14332
	Group B	14.79				
Time of full Motor recovery(mnts)	Group A	6.85	-1.946	<0.001	-2.574	-1.318
	Group B	8.79				

There were statistically significant differences in the duration of complete Analgesia, duration of effective analgesia and time of first pain medication between the study groups. All these parameters were longer in group B, compared to group A. (table 9)

Table 9:

Comparison of effectiveness of analgesia in both study groups (N=60)

Parameter	Group	Mean	Mean difference	P value	95% CI	
					Lower	Upper
Duration of complete Analgesia (VAS at 0)	Group A	8.92	-4.76029	<0.001	-5.38391	-4.13668
	Group B	13.69				
Duration of effective Analgesia (VAS at 4)	Group A	9.96	-4.693	<0.001	-5.395	-3.990
	Group B	14.65				
Time of First Pain Medication (VAS at 6)	Group A	12.00	-4.36731	<0.001	-5.11333	-3.62128
	Group B	16.36				

The average post anesthesia hemodynamic parameters were higher in group A compared to group B. The difference in the heart rate and diastolic blood pressure were statistically significant. The post anaesthetic respiratory parameters were almost similar in both the study groups. (Table 10)

Table 10:

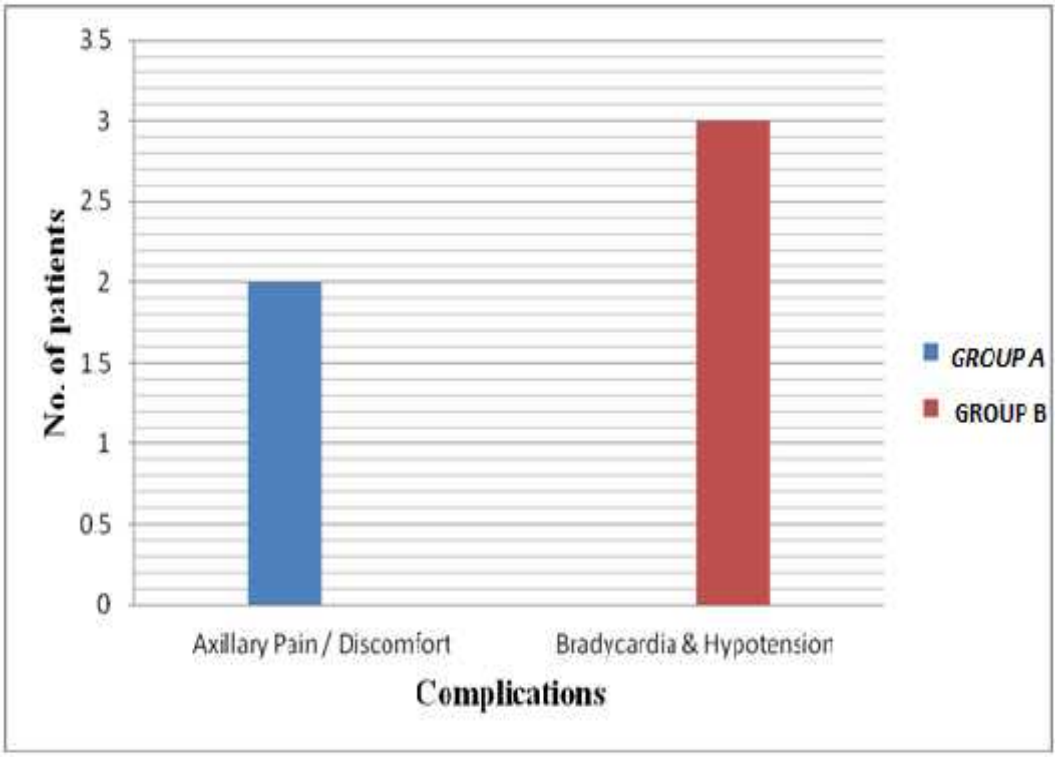
Comparison of Post anesthesia Hemodynamic Parameters (N=60)

Parameter	Group	Mean	Mean difference	P value	95% CI	
					Lower	Upper
Average Post Anesthesia Heart Rate	Group A	74.1814	3.540	0.026	0.431	6.651
	Group B	70.6410				
Average Post Anesthesia Systole	Group A	117.5392	2.632	0.315	-2.565	7.830
	Group B	114.9071				
Average Post Anesthesia Diastole	Group A	73.8480	3.576	0.014	0.745	6.406
	Group B	70.2724				
Average Post Anesthesia Respiratory rate	Group A	12.1299	-.00792	0.909	-.14632	.13048
	Group B	12.1378				
Average Post Anesthesia SPO2	Group A	99.9657	-.00867	0.762	-.06567	.04833
	Group B	99.9744				

Complications:

Complications	Group A	Group B
Axillary pain or discomfort	2	-
Bradycardia and	-	3

Axillary site pain and discomfort was complained by 2 patients in the group A whereas bradycardia and hypotension was observed in 3 patients in the group B which came to normal within 25minutes.



DISCUSSION

Regional Anesthesia is becoming more popular especially with the advent of safer drugs and techniques. Ultrasound has become more useful in the last few decades. Since both the drugs namely, levobupivacaine and dexmeditomidine are relatively newer in peripheral nerve block procedures, an attempt has been made to compare the two.

Until now, the common adjuvant used with local anesthetics was the opioids[57, 62]. More recently, α2 agonists have been used with good success. They improve the quality and duration of block in peripheral nerve blocks. The α2 agonists act through vasoconstriction, centrally acting pain relief, anti-inflammatory effects, hyperpolarization, decrease in compound action potential (CAP) and inhibition of voltage gate of sodium pump.

Age group:

Axillary block was conducted by Edson D. Carel in 1971 in pediatric age group by B. Fitz Gerald in 1976, by R.K. Mehta et al., in 1979, Blasier and White in 1996 and Colizza and Said in 1993, and supraclavicular brachial block in combination with general anesthesia was used for micro vascular surgery in children by Inberg P. et al., 1995.

In our study only adult patients were selected because of good patient cooperation with regard to the procedure.

Premedication

All the patients have received injection Midazolam 0.05mg/kg and injection Fentanyl 0.5µg/kg intravenously15 minutes before the procedure. This premedication was comparable with Sarita et al[22] where there was no premedication whereas Amany et al [21] have used midazolam of 1-2 mg and fentanyl 50 – 100 µg (not based on per kg dose) in all of their patients undergoing single shot infra clavicular block using bupivacaine with dexmeditomidine (2012).

Ultrasound

Andrea et al[27] compared the use of ultrasound and nerve stimulator for axillary block. They found that ultrasound has supremacy in 98.5% of successful blocks.

The findings of Vincent et al [28] and Christophe at al [29] also highlighted the role of ultrasound in the success of axillary block.

In our study, we observed that almost all the 60 patients had Grade 1 block (Good analgesia, sedatives were given only to relieve apprehension). This was made possible by the accuracy of the ultrasound in permitting direct visualization of the nerves[30].

Anaesthetic agent and dose

The recommended maximum dose for Levobupivacaine is 5mg/kg body weight in peripheral nerve blocks. This dose recommendation serves only as a base upon which a person using the drug in the technique should apply a sensible judgment and make appropriate adjustment.

Kenan Kaygusuz et al (2012)[62] and many others used 0.5% Levobupivacaine for axillary block (Paresthesia technique) with a dose of 200 mg (4 mg/kg) without any toxic symptoms.

In our study Levobupivacaine was used as an anaesthetic agent for all the cases in a concentration of 0.25%. We used Levobupivacaine in a dose of 2mg/kg body weight, with a total dose of only 100 mg, because of precise location of nerves made possible by ultrasound.

Onset of action

In a study of Sarita et al (2012)[22] where clonidine and Dexmeditomidine were compared in supraclavicular block, mean onset time of motor block in clonidine was 4.65 minutes whereas in Dexmeditomidine group was 3.87 minutes. The mean onset time of sensory block in clonidine group was 2.3 minutes whereas in Dexmeditomidine group was 1.7 minutes.

In a study by Kenan et al[63], when Dexmeditomedine was added with Levobupivacaine in axillary block, there was no shortening of onset of motor block whereas the onset of sensory block was shortened.

Keshav Govind Rao et al (2014)[63] and Rachana Gandhi et al[20] studied the effects of Dexmeditomidine with bupivacaine in supraclavicular block. They found that there was significant reduction of onset in the duration of motor and sensory blockade.

In our study the onset of sensory blockade (mean difference 0.04 minutes, p-value <0.001) and motor blockade (mean difference 0.03 minutes, p-value<0.001) were quicker in Levobupivacaine with

Dexmeditomidine group compared to plain Levobupivacaine group. Both these findings were statistically significant. The mean onset time of sensory block in plain Levobupivacaine was 7 minutes whereas in Levobupivacaine with Dexmeditomidine was 3 minutes. The mean onset time of motor block with plain Levobupivacaine was 10 minutes whereas in Levobupivacaine with Dexmeditomidine group was 7 minutes.

Thus, the durations of onset of sensory and motor block observed in our study are comparable with the above mentioned studies done earlier.

Duration of analgesia

Amany S. et al[21] compared bupivacaine alone and bupivacaine with Dexmeditomedine in ultrasound-guided single injection infraclavicular brachial plexus block. They reported that Dexmeditomidine group showed a statistically important reduced time of onset of sensory block (13.2 vs. 19.4 min, P=0.003), increased duration of the sensory block (179.4 vs. 122.7 min, P=0.002), reduced time of onset of motor block (15.3 vs. 22.2 min, P=0.003), prolonged duration of motor block (155.5 vs. 105.7 min, P=0.002), prolonged duration of postoperative analgesia (403 vs. 233 min, P=0.002) and reduced opioid requirements 48 hours after surgery.

Sarita et al[22], Kenan et al[63] and Aliye Esmaoglu et al[17] also reported similar effects in terms of prolongation of the duration of sensory and motor blocks.

In our study time taken for starting of regression (mean difference -1.37 minutes, p-value <0.108) was more in Levobupivacaine with

Dexmeditomedine compared to group levobupivacaine alone and this finding was statistically significant. There were statistically significant differences in the duration of complete analgesia, duration of effective analgesia and time of first pain medication between the study groups. All these three parameters were significantly prolonged in the group Levobupivacaine with Dexmeditomidine.

Quality of analgesia

Memis et al in their study have showed that when Dexmeditomedine was added with lignocaine for Biers block, it enhances the quality of analgesia.

In the study of Sarita et al, the quality of analgesia was 80% in patients with Dexmeditomedine whereas it was only 40% in patients with clonidine in supraclavicular block.

In our study, we graded the quality of analgesia into three grades and recorded the observations. Grade I analgesia was observed in 84.4% of patients in the levobupivacaine group whereas in the Levobupivacaine with Dexmeditomedine group, 93.6% of the patients were found to achieve grade I analgesia. The remaining patients in both the groups achieved Grade II analgesia.

Degree of motor blockade

Kenan et al observed that in both the groups with and without added Dexmeditomedine, 78% of patients achieved, Grade I degree of motor block.

In the present study, the degree of motor blockade observed in plain Levobupivacaine was found to be Grade I (Complete block, no active movements of entire elbow, forearm and hand) in 74.6% of patients and 86.6% in levobupivacaine with Dexmeditomidine patients. Grade II motor blockade was found to be 25.4% and 13.4% in Levobupivacaine group and Levobupivacaine with Dexmeditomedine group respectively.

These findings are comparable to the findings of the above mentioned study.

Hemodynamic Parameters

Esmaoglu et al[17] had observed bradycardia in their patient group in which 100 μg of Dexmeditomedine was used with Levobupivacaine.

In our study, our observations show that the hemodynamic parameters like heart rate and blood pressure were more in the optimal range in Levobupivacaine with Dexmeditomidine group than plain Levobupivacaine group. The respiratory parameters were almost similar in both the study groups. Bradycardia and hypotension (transient) were observed in 3 patients in the Levobupivacaine with Dexmeditomidine group.

The incidence of bradycardia was lesser in our study (only 3 cases) probably because of the lower dose of Dexmeditomidine we used. In our study we used 0.5 μg /kg of Dexmeditomidine with a maximum of 40 μg.

Complications

Vikram Uday Lahori and Anjana Raina et al (2011) have reported complications like accidental vascular puncture[45] in 2 patients of axillary block group.

In our study axillary pain or discomfort was the only complication in both the groups. No other complications or significant adverse effects were observed in both the study groups.

Summary

We conducted a prospective randomized double blinded case control study to compare Levobupivacaine and Levobupivacaine with Dexmeditomidine in ultrasound guided axillary block for elbow, forearm & hand surgeries. Sixty patients were divided into 2 groups of 30 patients each and axillary brachial plexus block with Levobupivacaine alone was performed on Group A patients and with Levobupivacaine and Dexmeditomidine on Group B patients. The patients were in the age group of 18 to 60 years of either sex, coming for surgeries, either emergency or planned, elbow & below the elbow joint in the orthopedics or plastic surgery department.

The drugs used were Levobupivacaine hydrochloride in a concentration of 0.25% and Dexmeditomidine at a dose of 0.5µg/kg. The concentration was fixed and dose and volume varied according to the body weight of the patient. Onset of analgesia was immediate with Dexmeditomidine with Levobupivacaine (3-5 minutes) whereas the onset of analgesia took 8-10 minutes in patients with Levobupivacaine alone. The quality of analgesia was Grade I in 76.6% patients with Levobupivacaine with Dexmeditomidine whereas it was Grade I in 50% patient's receiving

Levobupivacaine alone. Degree of motor block was grade I in 56.6% patients with Levobupivacaine with Dexmeditomidine as compared to only 26.6% patients with Levobupivacaine alone.

Duration of analgesia was found to be in the range of 8-10 hours with Levobupivacaine alone, whereas duration with 14-16 hours was found with Levobupivacaine with Dexmeditomidine.

Complications with both the drugs were found to be mild.

Conclusion

Based on our observations, we conclude that in ultrasound guided axillary block for elbow, forearm and hand surgeries, when compared to plain Levobupivacaine, the mixture of levobupivacaine with Dexmeditomidine produces

I. Statistically significant faster onset of sensory and motor blockade.

2. Statistically significant increase in duration of sensory and motor block.

3. Better grade of sensory and motor block, though this is not statistically significant.

4. without much increase in the incidence of complications.

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PROFORMA

USG guided Axillary Brachial Plexus block for elbow, forearm and Hand surgeries: A comparative study of Levobupivacaine & Levobupivacaine with Dexmeditomidine:

Name:

Age:

Sex:

IP No:

ASA Status: Height:

Weight:

Diagnosis:

Surgery:

Date of Surgery:

Anesthesiologist:

PRE-OP EVALUATION

GENERAL EXAMINATION

History:

Pallor: Icterus: Cyanosis: Clubbing:

Edema: Lymphadenopathy:

PR: BP:

RR:

SYSTEMIC EXAMINATION

CVS: CNS:

RS: P/A:

INVESTIGATIONS

Blood HB%:

FBS/RBS: Urea: Creatinine:

Chest X-Ray: ECG:

PREMEDICATION

ANAESTHETIC TECHNIQUE

USG Guided: 10 ml each for radial, ulnar, median, musculocutaneous nerve and medial cutaneous nerve of arm and forearm. 50 mm insulated needle used for injecting the LA solution.

Group A- 40 ml of 0.25% Levobupivacaine + 0.5 ml normal saline.

Group B- 40 ml of 0.25% Levobupivacaine + 0.5 ml Dexmeditomidine (0.5 ml= 25 µg)

MONITORING OF VITALS

Intra-op:

Time pre-op I mnt 3 5 10 15 20 25 30 45 60 120 150

HR

BP

RR

SPO2

SUPPLIMENTATION

OBSERVATIONS: Time of injection of drug in to the axillary sheath:

SENSORY BLOCKADE:

Time of onset (The time interval between administrations of local anesthetic solution to loss of pin prick sensation)

Duration of sensory block:

Duration of complete analgesia (Time of starting of regression / Return of pinprick sensation / VAS is >0)

Duration of effective analgesia (Time for full Sensory Recovery / VAS is >4):

Time to first pain medication (VAS>6):

MOTOR BLOCKADE:

Motor Blockade (Time of onset):

Motor Blockade (Degree/ Modified Bromage scale for upper extremities on a 3-point scale): Grade 1 /2 / 3

Grade 0: Normal motor function with full flexion and extension of elbow, wrist and fingers

Grade 1: Decreased motor strength with ability to move the fingers only

Grade 2: Complete motor block with inability to move the fingers

Motor Blockade (Duration of block):

Time for full motor recovery:

Quality of intra-op. analgesia (4 point modified Belzarena scale)

Grade 4: (Excellent) No complaint from patient

Grade 3: (Good) Minor complaint with no need for the supplemental analgesics

Grade 2: (Moderate) Complaint that required supplemental analgesia

Grade 1: (Unsuccessful) Patient given general anesthesia

Grade 1 / 2 / 3 / 4

Complications

(Bradycardia, hypotension, dizziness, nausea, vomiting, dryness of mouth, inadvertent vascular puncture etc.)

PATIENT CONSENT FORM

Study title: USG guided Axillary Brachial Plexus block for elbow, forearm and Hand surgeries A comparative study of Levobupivacaine & Levobupivacaine with Dexmeditomedine.

Study Centre: Department of Anesthesiology, GMKMCH, Salem.

Participant's Name: Age: IP NO:

The details of the study have been provided to me in writing and explained to me in my own language. I confirm that I have understood the above study and had the opportunity to ask questions. I understand that my participation in the study is voluntary and that I am free to withdraw at any time, without giving any reason, without my medical care or legal rights being affected. I agree not to restrict the use of any data or results that arise from this study provided such a use is only for scientific purposes. I fully consent to participate in the above study.

Signature of the participant with date:

Signature of the witness:

Signature &Name of the Anesthesiologist:

MASTER CHARTS

S.No	Name	Age	Sex	Group	IP No	ASA Status	Ht	Wt	Pre Op (HR)	1 mnt (HR)	3rd mnt (HR)	5th mnt (HR)	10th mnt (HR)	15 (HR)	20 (HR)
1	Sundhari	55	Female	Group B	66582	1	1.6	70	118	112	95	95	72	70	66
2	Murugan	24	Male	Group B	56530	1	1.76	70	76	74	72	68	68	70	72
3	Govindhan	55	Male	Group A	59154	2	1.7	75	90	88	80	76	72	74	70
4	Pavunu	50	Female	Group A	71024	2	1.55	90	88	76	78	74	70	68	67
5	Govindhan	18	Male	Group B	70294	1	1.7	65	96	88	74	72	73	72	72
6	Babu	45	Male	Group B	68928	1	1.7	80	84	82	76	74	73	72	72
7	Ramesh	27	Male	Group A	70398	1	1.74	70	88	79	72	74	70	73	72
8	Rajeswari	36	Male	Group A	69080	1	1.68	60	82	78	77	78	76	72	72
9	Kumar	30	Male	Group B	69320	1	1.72	70	96	88	74	72	72	76	76
10	Ellappan	55	Male	Group A	69080	1	1.67	60	86	84	78	78	84	77	76
11	KulanthaiVadivel.	58	Male	Group A	70022	2	1.65	78	66	89	78	72	70	68	68
12	Thulasiraman	18	Male	Group B	69874	1	1.7	60	102	96	84	78	72	72	76
13	chandraleka	19	Female	Group B	73900	1	1.5	40	148	136	124	118	84	78	82
14	SundaraMoorthy	53	Male	Group B	68920	1	1.67	70	68	68	66	61	58	57	57
15	Mayilsamy	31	Male	Group B	69572	1	1.65	75	68	86	74	68	66	59	54
16	Nithya	30	Female	Group B	67810	1	1.6	55	92	92	90	84	78	76	75
17	Subramanyan	40	Male	Group A	52524	2	1.7	70	68	68	72	66	68	64	66
18	Kuppayee	50	Female	Group A	64550	2	1.58	60	88	83	86	94	102	81	72
19	Kanjamalai	50	Male	Group A	18782	1	1.65	74	62	63	62	66	62	64	59
20	Dhanam	40	Female	Group B	69136	2	1.55	70	79	78	74	69	66	66	68
21	Kamala	25	Female	Group B	53582	1	1.6	55	100	98	98	87	88	73	68
22	Vijayakumar	31	Male	Group A	38128-OP	1	1.75	80	88	86	82	78	77	77	77
23	Gowtham	18	Male	Group A	70294	1	1.76	60	78	78	77	78	75	76	77
24	Devan	54	Male	Group A	73056	2	1.6	70	98	93	94	86	85	82	80
25	Saraswathy	48	Female	Group A	42986	2- DM	1.55	70	78	78	77	75	76	75	74
26	Kumar	45	Male	Group A	72720	1	1.68	75	96	92	88	86	87	84	82
27	subramani	50	Male	Group B	59088	1	1.73	55	94	86	77	78	74	68	72
28	Venkatesh	49	Male	Group A	47126-OP	1	1.68	70	82	82	80	78	77	81	73
29	Murugan raj	29	Male	Group B	62244	1	1.76	75	78	77	72	66	65	62	58
30	Anbarasu	22	Male	Group A	38028	1	1.7	62	58	58	57	60	58	59	60
31	Priya	19	Female	Group A	39844	1	1.65	53	92	91	88	86	82	78	76
32	Utherasamy	48	Male	Group B	30798	2	1.78	64	78	78	77	76	68	62	64
33	Ganeshan	35	Male	Group B	54574	1	1.74	75	78	76	72	78	80	72	68
34	Thana kodi	26	Female	Group A	57576	2	1.6	50	92	85	78	76	76	75	74
35	Madhu	50	Male	Group B	49690	1	1.75	80	68	67	68	62	58	57	58
36	Selvi	55	Female	Group A	71050	2	1.55	70	78	77	78	81	73	71	68
37	Santha	58	Female	Group A	27810	1	1.5	60	76	76	74	73	76	72	72
38	Balu	26	Male	Group B	49954	1	1.75	70	82	80	76	78	74	71	62
39	Nallakodi	46	Male	Group A	36706	1	1.7	80	78	76	76	75	72	72	72
40	Kamala	25	Female	Group A	53582	1	1.68	60	90	88	86	85	78	74	72
41	Sakthivel	19	Male	Group A	58428	1	1.76	55	88	82	78	78	76	72	70
42	Indhumathi	19	Female	Group B	70262	1	1.6	45	108	92	86	75	76	68	72
43	Narayanan	55	Male	Group B	43490	2	1.8	85	72	70	68	66	65	68	68
44	Chinnaponnu	32	Female	Group B	54142	2	1.58	70	102	98	92	86	77	74	73
45	vasanth	24	Male	Group A	50720	1	1.75	70	82	81	77	72	66	63	68
46	saminathan	39	Male	Group A	43602	1	1.74	85	66	66	73	72	72	76	75
47	Gopal	44	Male	Group B	57720	1	1.72	78	88	87	85	81	77	75	72
48	Sathishkumar	25	Male	Group A	63306	1	1.7	65	86	86	85	82	81	78	76
49	Balamurugan	26	Male	Group A	70662	1	1.7	75	110	108	104	98	97	95	92
50	Nethaji	58	Male	Group A	69876	2	1.72	78	68	68	67	66	67	65	64
51	Sengottaiyan	42	Male	Group A	44922	1	1.72	68	78	78	77	78	76	75	76
52	Harish	18	Male	Group A	43446	1	1.68	55	90	88	89	87	82	78	76
53	Manian	22	Male	Group A	44100	1	1.76	62	82	82	80	76	78	78	76
54	sathish	19	male	Group B	32430	1	1.78	63	82	82	80	78	72	74	72
55	Jeyaprakash	26	Male	Group B	41564	1	1.75	79	78	78	76	77	78	74	64
56	Natesan	18	Male	Group A	41513	1	1.68	55	83	83	82	78	75	76	72
57	Mani	45	Male	Group B	23536	2	1.65	80	68	68	62	58	53	48	48
58	Naveen Kumar	18	Male	Group A	27075	1	1.7	58	90	89	85	83	78	77	74
59	Govind	48	Male	Group A	31458	1	1.64	75	68	68	70	67	66	68	67
60	Malarkodi	23	Female	Group B	31017	1	1.65	65	87	86	84	78	77	73	72

Sl. No	25 (HR)	30 (HR)	45 (HR)	60 (HR)	120 (HR)	150 (HR)	Pre Op (Systole)	Pre Op (Diastole)	1 mnt (Systole)	1 mnt (Diastole)	3rd mnt (Systole)	3rd mnt (Diastole)	5th mnt (Systole)	5th mnt (Diastole)	10th mnt (Systole)	10th mnt (Diastole)	15 (Systole)	15 (Diastole)	20 (Systole)	20 (Diastole)	25 (Systole)	25 (Diastole)
1	54	48	49	55	56	55	108	69	124	92	119	66	102	58	108	62	94	45	112	68	110	65
2	74	72	74	72	74	72	116	77	108	69	110	68	102	66	108	69	112	66	112	66	110	70
3	72	74	76	72	73	76	160	84	154	78	137	69	134	66	129	67	132	69	129	68	118	55
4	68	69	65	62	74	69	186	78	168	83	206	79	186	79	153	61	142	55	146	66	155	68
5	74	70	72	69	66	74	128	92	126	87	108	66	106	64	108	66	104	58	106	59	102	48
6	69	73	66	68	78	74	158	96	148	88	136	72	127	68	122	66	135	69	108	54	121	67
7	76	73	68	69	70	74	134	89	122	78	118	70	114	68	108	66	110	69	98	54	106	66
8	74	86	77	73	72	70	138	86	129	76	122	69	111	70	104	68	106	69	109	72	118	66
9	75	72	72	69	72	72	138	96	126	82	118	67	109	62	116	68	108	59	112	65	102	54
10	72	69	72	73	78	82	134	98	136	100	128	96	116	82	115	84	108	78	110	70	108	66
11	66	64	57	56	62	62	168	97	176	102	158	96	144	82	146	83	142	81	138	64	136	63
12	78	68	66	70	72	68	130	90	128	86	118	82	110	76	106	75	102	74	92	58	102	66
13	78	66	63	58	64	72	138	100	126	88	120	86	118	79	110	68	102	59	106	58	110	66
14	55	57	58	62	59	62	132	89	128	82	127	82	121	79	116	77	115	77	114	78	107	73
15	54	56	52	58	60	62	132	88	128	86	118	78	106	66	105	66	97	58	102	58	106	60
16	72	68	66	67	68	72	138	77	137	75	133	74	129	72	124	69	122	68	118	66	113	67
17	64	62	58	62	63	63	136	98	128	84	126	83	122	82	122	82	116	78	108	76	112	81
18	68	70	74	72	74	72	138	86	129	84	118	78	110	70	103	69	104	71	122	82	113	76
19	54	60	62	66	64	62	140	90	146	108	138	96	124	87	123	88	121	82	108	67	100	61
20	65	65	68	65	65	69	184	92	144	74	139	72	143	77	148	78	142	78	152	78	157	78
21	68	66	62	58	63	68	140	90	138	88	134	86	122	78	121	78	108	69	114	72	107	70
22	75	74	75	76	74	72	138	88	129	84	128	84	126	87	127	88	119	83	119	84	109	78
23	74	74	78	84	86	78	120	80	118	82	117	81	114	78	106	71	101	66	121	82	123	82
24	78	77	74	73	72	72	160	100	148	96	146	95	140	88	138	82	126	80	125	82	118	80
25	77	73	72	68	70	72	130	90	128	88	127	88	122	86	124	85	127	87	122	88	116	82
26	83	82	83	81	80	76	170	100	168	99	166	98	166	98	163	88	158	84	147	78	144	76
27	70	68	66	70	64	68	108	76	98	69	84	56	102	66	97	63	105	66	114	72	122	78
28	74	76	74	77	78	76	130	80	128	80	126	77	122	76	118	72	117	73	106	58	102	55
29	57	58	62	62	66	68	134	86	128	83	117	78	116	77	109	68	102	64	98	59	108	62
30	62	66	58	56	60	62	108	66	108	65	104	62	102	58	108	64	115	71	110	68	97	54
31	76	74	73	73	68	72	129	87	129	87	123	82	118	79	121	78	117	78	112	76	110	75
32	66	68	74	72	73	75	108	72	102	72	94	65	98	67	102	68	91	59	95	63	97	65
33	66	68	70	72	77	72	124	86	112	78	102	66	98	62	104	68	110	72	118	76	122	75
34	74	73	72	68	70	72	108	66	110	68	114	73	107	76	113	82	114	78	112	73	108	66
35	58	62	68	70	76	72	140	90	138	87	125	78	122	76	104	67	87	58	101	64	108	69
36	66	69	72	72	73	74	150	100	146	98	145	98	140	92	140	92	134	86	133	87	129	94
37	74	69	70	68	76	82	110	70	108	68	102	61	93	54	89	48	94	52	102	58	105	62
38	66	58	63	59	65	67	124	82	123	82	120	81	118	78	106	63	101	58	98	54	106	60
39	73	76	74	78	72	76	138	92	122	78	121	77	128	82	126	80	118	78	116	77	114	75
40	73	76	74	73	72	72	130	90	128	89	122	84	120	86	114	78	106	76	102	72	110	78
41	72	74	77	73	72	72	110	70	109	68	106	66	103	62	94	58	114	66	110	70	106	67
42	69	68	68	64	68	68	150	97	136	82	129	82	128	81	126	80	106	68	98	67	86	58
43	70	72	68	66	70	72	150	100	148	96	144	92	142	88	138	84	126	78	125	77	121	73
44	72	72	68	67	68	76	148	93	148	93	144	87	132	78	128	73	109	64	92	58	98	66
45	66	62	66	68	72	74	121	77	118	76	109	72	101	71	87	59	96	68	103	69	110	70
46	77	72	70	72	74	76	138	89	137	86	133	84	128	83	122	78	123	78	119	76	120	76
47	68	67	65	68	67	70	130	70	128	66	122	65	108	67	98	54	102	58	108	62	112	66
48	75	72	77	72	73	74	110	70	110	68	108	67	106	66	108	69	110	72	108	76	112	78
49	84	82	76	72	73	73	128	86	126	85	122	82	116	78	108	77	106	76	102	68	98	64
50	68	65	63	62	62	64	154	98	148	93	148	94	144	89	138	83	129	76	128	77	126	75
51	74	72	73	74	72	73	126	77	122	76	121	76	118	74	108	75	102	72	97	66	91	58
52	74	72	72	73	74	72	110	70	108	71	104	68	100	66	96	54	98	52	102	58	108	60
53	77	78	72	74	72	73	116	68	115	68	114	70	115	72	108	69	112	72	108	66	110	69
54	77	74	68	72	71	74	108	72	106	73	102	72	90	59	92	60	100	66	103	75	106	77
55	62	58	62	66	62	68	118	76	121	72	116	68	114	66	110	62	104	58	93	48	92	42
56	73	76	78	73	76	74	128	92	129	93	122	86	114	77	108	68	103	63	98	59	112	63
57	50	46	42	44	56	64	146	87	145	84	138	77	129	72	121	68	108	57	98	53	100	55
58	74	73	72	72	76	74	110	70	108	70	105	68	103	67	91	58	94	60	102	63	102	63
59	69	66	68	70	72	70	132	88	128	87	125	88	128	86	123	82	116	76	112	77	108	78
60	68	67	63	68	68	76	137	92	136	91	132	88	129	87	128	85	127	83	121	78	117	77

Sl. No	30 (Systole)	30 (Diastole)	45 (Systole)	45 (Diastole)	60 (Systole)	60 (Diastole)	120 (Systole)	120 (Diastole)	150 (Systole)	150 (Diastole)	Pre Op(RR)	1 mnt (RR)	3rd mnt (RR)	5th mnt (RR)	10th mnt (RR)	15 (RR)	20 (RR)	25 (RR)	30 (RR)	45 (RR)	60 (RR)	120 (RR)
1	109	68	121	70	104	63	110	70	132	89	14	12	12	12	12	12	12	12	12	12	12	12
2	114	72	112	68	108	66	110	72	108	79	15	12	12	12	12	12	12	12	12	12	12	12
3	120	60	118	64	128	69	135	71	133	68	16	14	12	12	12	12	12	12	12	12	12	12
4	124	56	126	58	122	55	136	62	128	66	12	12	12	12	12	12	12	12	12	12	12	12
5	98	47	107	65	112	68	102	62	112	66	14	12	12	12	12	12	12	12	12	12	12	12
6	112	62	128	72	136	81	128	76	122	71	14	12	12	12	12	12	12	12	12	12	12	12
7	112	67	118	74	110	70	108	66	114	78	12	12	12	12	12	12	12	12	12	12	12	12
8	124	71	110	72	102	68	110	69	119	72	14	12	12	12	12	12	12	12	12	12	12	12
9	108	64	114	66	101	62	118	75	116	74	14	12	12	12	12	12	12	12	12	12	12	12
10	106	68	102	65	98	66	106	70	116	72	14	12	12	12	12	12	12	12	12	12	12	12
11	127	62	128	66	122	64	126	68	127	73	14	14	12	12	12	12	12	12	12	12	12	12
12	98	65	104	67	112	72	114	78	121	76	16	14	12	12	12	12	12	12	12	12	12	12
13	104	55	101	54	93	62	106	68	112	70	20	18	16	12	12	12	12	12	12	12	12	12
14	108	77	103	75	110	81	112	82	124	79	12	12	12	12	12	12	12	12	12	12	12	12
15	112	66	134	69	127	73	128	74	117	66	12	12	12	12	12	12	12	12	12	12	12	12
16	109	58	101	56	117	63	121	68	128	73	14	12	12	12	12	12	12	12	12	12	12	12
17	110	74	106	72	112	76	125	86	122	83	12	12	12	12	12	12	12	12	12	12	12	12
18	108	72	98	67	104	77	104	78	118	82	14	12	12	12	12	12	12	12	12	12	12	12
19	116	87	102	63	92	58	102	66	126	72	14	12	12	12	12	12	12	12	12	12	12	12
20	160	82	146	76	128	69	127	68	131	70	16	14	12	12	12	12	12	12	12	12	12	12
21	105	68	97	58	109	67	114	68	121	66	16	16	14	12	12	12	12	12	12	12	12	12
22	108	79	102	71	98	68	106	74	110	75	12	12	12	12	12	12	12	12	12	12	12	12
23	118	79	117	82	116	78	114	77	117	75	14	12	12	12	12	12	12	12	12	12	12	12
24	109	76	110	77	117	79	123	81	124	83	14	12	12	12	12	12	12	12	12	12	12	12
25	108	78	102	76	110	79	117	82	136	83	16	16	14	12	12	12	12	12	12	12	12	12
26	138	69	128	66	118	67	127	71	132	72	16	16	14	14	12	12	12	12	12	12	12	12
27	118	73	116	69	117	75	118	79	109	66	16	14	12	12	12	12	12	12	12	12	12	12
28	116	62	112	59	118	63	119	65	110	66	12	12	12	12	12	12	12	12	12	12	12	12
29	112	66	118	68	132	75	131	73	128	74	14	12	12	12	12	12	12	12	12	12	12	12
30	108	62	105	63	106	65	107	68	110	72	12	12	12	12	12	12	12	12	12	12	12	12
31	108	77	107	78	110	76	112	69	108	66	16	16	16	14	12	12	12	12	12	12	12	12
32	104	68	110	72	112	73	116	78	118	82	12	12	12	12	12	12	12	12	12	12	12	12
33	116	72	118	74	126	81	127	82	118	77	12	12	12	12	12	12	12	12	12	12	12	12
34	106	64	110	68	102	66	116	73	124	82	16	14	12	12	12	12	12	12	12	12	12	12
35	114	66	126	72	125	73	107	68	102	66	12	12	12	12	12	12	12	12	12	12	12	12
36	129	76	128	76	121	72	118	68	119	72	14	12	12	12	12	12	12	12	12	12	12	12
37	110	72	107	68	103	69	104	73	121	76	12	12	12	12	12	12	12	12	12	12	12	12
38	113	68	110	66	109	66	117	71	120	76	12	12	12	12	12	12	12	12	12	12	12	12
39	113	77	114	75	116	78	113	78	120	83	12	12	12	12	12	12	12	12	12	12	12	12
40	110	79	108	76	110	78	122	84	120	81	14	14	14	12	12	12	12	12	12	12	12	12
41	108	72	109	74	114	74	120	78	126	83	16	16	16	14	12	12	12	12	12	12	12	12
42	97	69	100	70	102	71	112	76	116	77	18	16	15	14	12	12	12	12	12	12	12	12
43	118	74	116	69	104	58	106	60	108	66	12	12	12	12	12	12	12	12	12	12	12	12
44	102	67	118	73	119	73	125	78	128	80	16	12	12	12	12	12	12	12	12	12	12	12
45	116	73	127	75	118	76	119	77	121	76	12	12	12	12	12	12	12	12	12	12	12	12
46	114	73	113	76	110	72	118	74	124	77	12	12	12	12	12	12	12	12	12	12	12	12
47	114	68	121	72	120	72	127	78	129	74	12	12	12	12	12	12	12	12	12	12	12	12
48	114	77	116	78	118	79	117	76	118	75	12	12	12	12	12	12	12	12	12	12	12	12
49	107	68	110	69	118	72	120	73	122	78	14	12	12	12	12	12	12	12	12	12	12	12
50	117	71	118	74	121	81	124	83	128	86	12	12	12	12	12	12	12	12	12	12	12	12
51	108	63	112	68	110	70	112	67	116	68	12	12	12	12	12	12	12	12	12	12	12	12
52	112	73	110	72	114	75	116	72	122	78	12	12	12	12	12	12	12	12	12	12	12	12
53	104	58	100	56	111	65	110	67	112	68	12	12	12	12	12	12	12	12	12	12	12	12
54	102	72	104	73	105	72	116	83	118	86	12	12	12	12	12	12	12	12	12	12	12	12
55	96	52	101	55	110	63	123	66	128	64	12	12	12	12	12	12	12	12	12	12	12	12
56	104	66	110	68	104	56	100	58	108	66	16	14	14	12	12	12	12	12	12	12	12	12
57	98	52	110	58	118	63	124	66	128	72	14	12	12	12	12	12	12	12	12	12	12	12
58	106	68	112	70	114	72	115	73	116	71	14	14	14	13	12	12	12	12	12	12	12	12
59	99	67	102	68	106	68	114	66	118	70	12	12	12	12	12	12	12	12	12	12	12	12
60	114	75	118	76	109	68	102	62	116	71	16	16	16	14	14	12	12	12	12	12	12	12

Sl. No	3rd mnt (SPO2)	5th mnt (SPO2)	10th mnt (SPO2)	15 (SPO2)	20 (SPO2)	25 (SPO2)	30 (SPO2)	45 (SPO2)	60 (SPO2)	120 (SPO2)	150(SPO2)	Time of injection of drug in to the axillary sheath	Time of onset	Time of starting of regression	Duration of sensory block { Return of pinprick sensation}:	Motor Blockade (Time of onset)	Motor Blockade (Duration of block)	Time for full Sensory Recovery { mnts }:	Time for full motor recovery (mnts)	Duration of complete analgesia { VAS is >0 }	Duration of effective analgesia { VAS is >4 }	Time to first pain medication { VAS >6 }
1	99	100	100	100	100	100	100	100	100	100	100	08.00 pm	08.06 pm	10.50 am	14.44	8.10 pm	6.1	15.55	10	14.44	15.15	15.55
2	100	100	100	100	100	100	100	100	100	100	100	09.00 am	09.04 am	10.30 am	13.26	09.10 am	10.3	14.26	10.3	13.26	13.45	16
3	99	100	100	100	100	100	100	100	100	100	100	11.15 am	11.21 am	07.21 pm	8	11.32 am	6.15	9.15	7.15	8	8.3	10
4	98	99	100	100	100	100	100	100	100	100	100	09.10 am	09.16 am	05.00 pm	7.44	9.46 am	5.45	9	6	8	8.2	10
5	100	100	100	100	100	100	100	100	100	100	100	11.10 am	11.04 am	01 30 am	14.26	11.10 am	10	16.1	14	16.1	15.3	18
6	100	100	100	100	100	100	100	100	100	100	100	08.50 am	08.54am	10.50 pm	13.56	09.00 am	10.1	14.56	10.4	13.56	14.25	16
7	100	100	100	100	100	100	100	100	100	100	100	11.00 am	11.08 am	09.00 pm	9.52	11.15 am	6.3	10.3	7	9.52	10	12
8	100	100	100	100	100	100	100	100	100	100	100	09.30 am	09.38am	06.10 pm	8.32	09.55 am	5.15	9.32	6	8.32	9	10
9	100	100	100	100	100	100	100	100	100	100	100	10.50 am	10.53 am	11.30 pm	12.37	11.05 am	8.3	13.15	9	12.37	12.55	15
10	100	100	100	100	100	100	100	100	100	100	100	09.00 am	09.08 am	06.15 pm	9.07	9.15	5.3	10.07	6.3	9.07	9.45	10
11	99	100	100	100	100	100	100	100	100	100	100	09.15 am	09.20 am	05.20 pm	8	9.28	5.15	9.05	6	8	8.45	10
12	100	100	100	100	100	100	100	100	100	100	100	10.40 am	10.44 am	01.00 am	14.16	10.48	9.16	16	10	14.16	15.3	16
13	100	100	100	100	100	100	100	100	100	100	100	09.00 am	09.04 am	11.15 pm	14.11	09.12 am	8.15	15.3	9	14.11	14.45	16
14	100	100	100	100	100	100	100	100	100	100	100	11.00 am	11.04 am	01.30 am of next day	14.26	11.12 am	8.15	15.3	9.3	14.26	15.3	18
15	100	100	100	100	100	100	100	100	100	100	100	12.15 pm	12.18 pm	01.30 am of next day	13.12	12.26 pm	7.15	14.3	8.3	13.13	15.5	18
16	100	100	100	100	100	100	100	100	100	100	100	10.30 am	10.33 am	12.50 am of next day	14.17	10.38 am	7.4	15.3	8.3	14.17	15.15	16
17	100	100	100	100	100	100	100	100	100	100	100	12.15 pm	12.19 pm	09.45 pm	9.26	12.25 pm	6.3	10.5	7.15	9.26	10.3	12
18	100	100	100	100	100	100	100	100	100	100	100	09.10 am	09.18 am	06.18 pm	9	05.45 hrs	6.3	10.15	7	9	9.45	12
19	100	100	100	100	100	100	100	100	100	100	100	11.00 am	11.08 am	08.45 pm	9.37	11.15 am	6.3	10.15	7.3	9.37	9.5	12
20	99	100	100	100	100	100	100	100	100	100	100	11.20 am	11.25 am	02.00 am of 23.8.14	14.35	11.35 am	10.15	15.35	11	14.35	15	18
21	100	100	100	100	100	100	100	100	100	100	100	10.30 am	10.33 am	12.33 am of next day.	14	10.42 am	7.4	15.4	8.3	14	16.3	18
22	100	100	100	100	100	100	100	100	100	100	100	11.50 am	11.58 am	10.30 pm	11.28	12.03 pm	7.4	12.5	8.3	11.28	12.45	14
23	100	100	100	100	100	100	100	100	100	100	100	09.00 am	09.05 am	10.15 pm	12.1	09.10 am	6.1	13.2	7	12.1	14 .00	16
24	100	100	100	100	100	100	100	100	100	100	100	05.30 pm	05.38 pm	02.30 am	8.52	05.41 pm	6.2	10.2	7.1	8.52	10	12
25	100	100	100	100	100	100	100	100	100	100	100	08.50 am	08.58 am	05.00 pm	8.02	09.04 am	5.5	9.5	7	8.02	9.3	12
26	100	100	100	100	100	100	100	100	100	100	100	09.00 am	09.08 am	05.00 pm	7.58	09.14 am	5.15	8.5	6	7.58	8.45	10
27	100	100	100	100	100	100	100	100	100	100	100	08.50 am	08.53 am	10.53 pm	14	08.58 pm	7.2	15.1	8.3	14	15.1	16
28	100	100	100	100	100	100	100	100	100	100	100	12.30 pm	12.37 pm	11.15 pm	10.38	12.40 pm	6.3	11.5	7.15	10.38	12	13
29	100	100	100	100	100	100	100	100	100	100	100	10.40 am	10.44 am	12.15 am of next day.	11.29	10.41 am	5.5	12.45	7	11.29	12.1	14
30	100	100	100	100	100	100	100	100	100	100	100	10.30 am	10.37 am	06.00 pm	7.23	10.43 am	4.55	8.5	6	7.23	8.3	10

Sl. No	3rd mnt (SPO2)	5th mnt (SPO2)	10th mnt (SPO2)	15 (SPO2)	20 (SPO2)	25 (SPO2)	30 (SPO2)	45 (SPO2)	60 (SPO2)	120 (SPO2)	150(SPO2)	Time of injection of drug in to the axillary sheath	Time of onset	Time of starting of regression	Duration of sensory block{ Return of pinprick sensation}:	Motor Blockade(Time of onset)	Motor Blockade(Duration of block)	Time for full Sensory Recovery{ mnts }:	Time for full motor recovery(mnts)	Duration of complete analgesia{ VAS is >0 }	Duration of effective analgesia { VAS is >4 }	Time to first pain medication{ VAS >6 }
31	100	100	100	100	100	100	100	100	100	100	100	11.40 am	11.47 am	08.15 pm	8.28	11.52 am	5.3	9.4	06 15	8.28	9.15	11
32	100	100	100	100	100	100	100	100	100	100	100	10.30 am	10.33 am	12.40 am of next day	15.07	10.38 am	6.4	16.15	8	15.07	16	17
33	100	100	100	100	100	100	100	100	100	100	100	11.30 am	11.34 am	12.50 am of next day.	13.16	11.44 am	7.1	15.16	8	13.16	14.3	16
34	100	100	100	100	100	100	100	100	100	100	100	12.15 pm	12.24 pm	09.30 pm	9.06	12.30 pm	6.3	7.15	10.15	9.06	10.3	1
35	100	100	100	100	100	100	100	100	100	100	100	08.50 am	08.54 am	10.30 pm	13.36	09.03 am	5.5	14.4	7.1	13.36	14.45	16
36	100	100	100	100	100	100	100	100	100	100	100	11.20 am	11.28 am	07.28 pm	9	11.40 am	6.15	10.15	7	9	9.4	14
37	100	100	100	100	100	100	100	100	100	100	100	12.15 pm	12.24 pm	09.30 pm	9.06	12.32 am	5.15	10.3	6	9.06	9.5	12
38	100	100	100	100	100	100	100	100	100	100	100	10.40 am	10.44 am	12.44 am of next day.	14	10.58 am	7.3	15.15	8.3	14	14.45	17
39	100	100	100	100	100	100	100	100	100	100	100	11.30 am	11.37 am	09.00 pm	9.23	11.40 am	6.4	10.3	7.3	9.23	10	12
40	100	100	100	100	100	100	100	100	100	100	100	11.20 am	11.28 am	10.00 pm	10.32	11.40 am	6.1	11.2	7	10.32	11.05	14
41	100	100	100	100	100	100	100	100	100	100	100	12.30 pm	12.38 pm	07.40 pm	8.02	12.42 pm	6.4	9	7	8.02	9	12
42	100	100	100	100	100	100	100	100	100	100	100	10.15 am	10.18 am	11.45 pm	13.27	10.30 am	8.3	14.5	9.3	13.27	14.05	17
43	100	100	100	100	100	100	100	100	100	100	100	09.00 am	09.04 am	11.04 pm	14	09.10 am	6.4	14.5	7.15	14	15.15	16
44	100	100	100	100	100	100	100	100	100	100	100	08.50 am	08.53 am	10.30 pm	13.37	09.00 am		14.4	7.15	13.37	14.5	16
45	100	100	100	100	100	100	100	100	100	100	100	10.45 am	10.49 am	10.30 pm	11.41	10.56 am	5.45	12.5	6.4	11.41	13.3	15
46	100	100	100	100	100	100	100	100	100	100	100	11.10 am	11.18 am	12.30 am of next day	13.12	11.20 hrs	7.1	14.3	8.15	13.12	14.45	16
47	100	100	100	100	100	100	100	100	100	100	100	12.30 pm	12.34 pm	01.30 am	12.56	12.39 pm	6.3	13.5	7.4	12.56	14	16
48	100	100	100	100	100	100	100	100	100	100	100	02.50 pm	02.57 pm	11.30 pm	8.33	3.02 pm	5.15	9.4	6.1	8.33	9.5	12
49	100	100	100	100	100	100	100	100	100	100	100	10.30 am	10.37 am	07.30 pm	8.53	10.43 am	6.45	10.3	7.4	8.53	10.3	12
50	100	100	100	100	100	100	100	100	100	100	100	11.30 am	11.37 am	08.00 pm	8.23	11.49 am	5.4	10	6.3	8.23	10.15	13
51	100	100	100	100	100	100	100	100	100	100	100	11.50 am	11.57 am	08.00 pm	8.03	12.00 am	5.5	9.4	6.4	8.03	9.5	12
52	100	100	100	100	100	100	100	100	100	100	100	08.50 am	08.56 am	05.15 pm	8.19	09.00 am	5.3	9.35	6.2	8.19	9.2	12
53	100	100	100	100	100	100	100	100	100	100	100	12.20 pm	12.28 pm	07.30 pm	8.02	12.29 pm	6.5	9.3	7.5	8.02	10	12
54	100	100	100	100	100	100	100	100	100	100	100	10.20 am	10.24 am	11.30 pm	13.06	10.28 am	7.05	14.5	8	13.06	14.4	16
55	100	100	100	100	100	100	100	100	100	100	100	10.30 am	10.33 am	12.40 am of next	14.07	10.38 am	7.3	15.4	8.4	14.07	16	17

														day							
56	100	100	100	100	100	100	100	100	100	100	100	12.30 pm	12.38 pm	08.38 pm	8	12.42 pm	5.15	9.15	6.2	8	9.3	12
57	100	100	100	100	100	100	100	100	100	100	100	08.50 am	08.54 am	10.30 pm	13.36	08.58 am	7.4	14.5	8.5	13.36	14.4	15
58	100	100	100	100	100	100	100	100	100	100	100	08.50 am	08.59 am	05.40 pm	8.01	09.05 am	5.05	9.2	6	8.01	9	10
59	100	100	100	100	100	100	100	100	100	100	100	10.40 am	10.48 am	06.30 pm	7.12	10.53 am	5.5	8.4	6.45	7.12	8.3	10
60	100	100	100	100	100	100	100	100	100	100	100	10.30 am	10.34 am	12.20 am of next day	13.46	10.38 am	7.15	14.4	8.15	13.46	14.3	16

Fig.1 Formation of brachial plexus

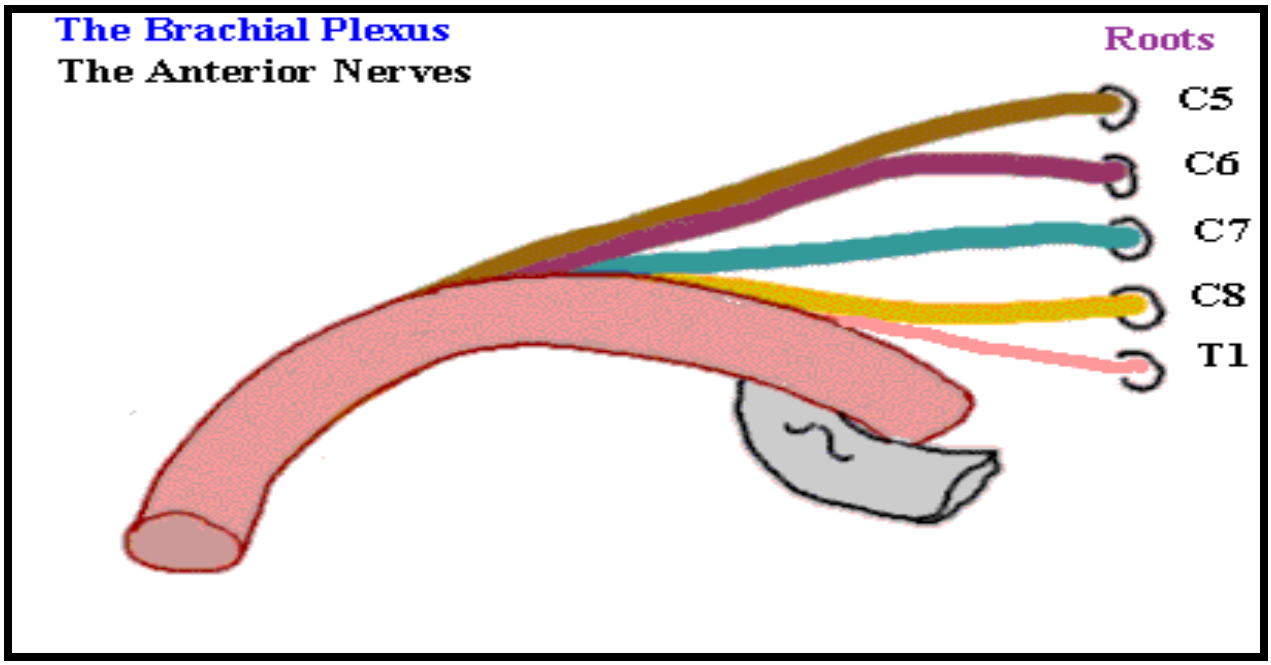

Fig.2 Anatomy of Brachial Plexus

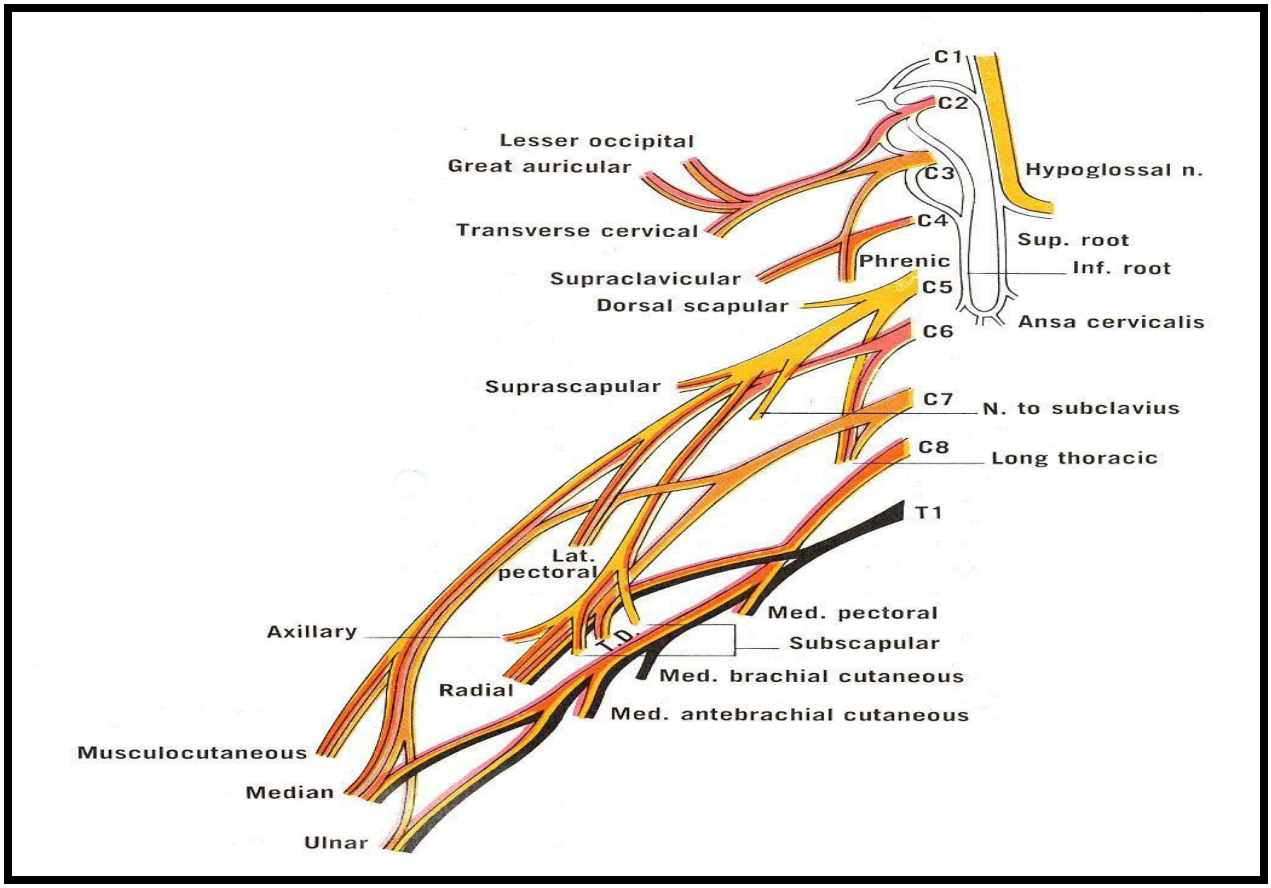

Fig.3 Structure of Brachial Plexus

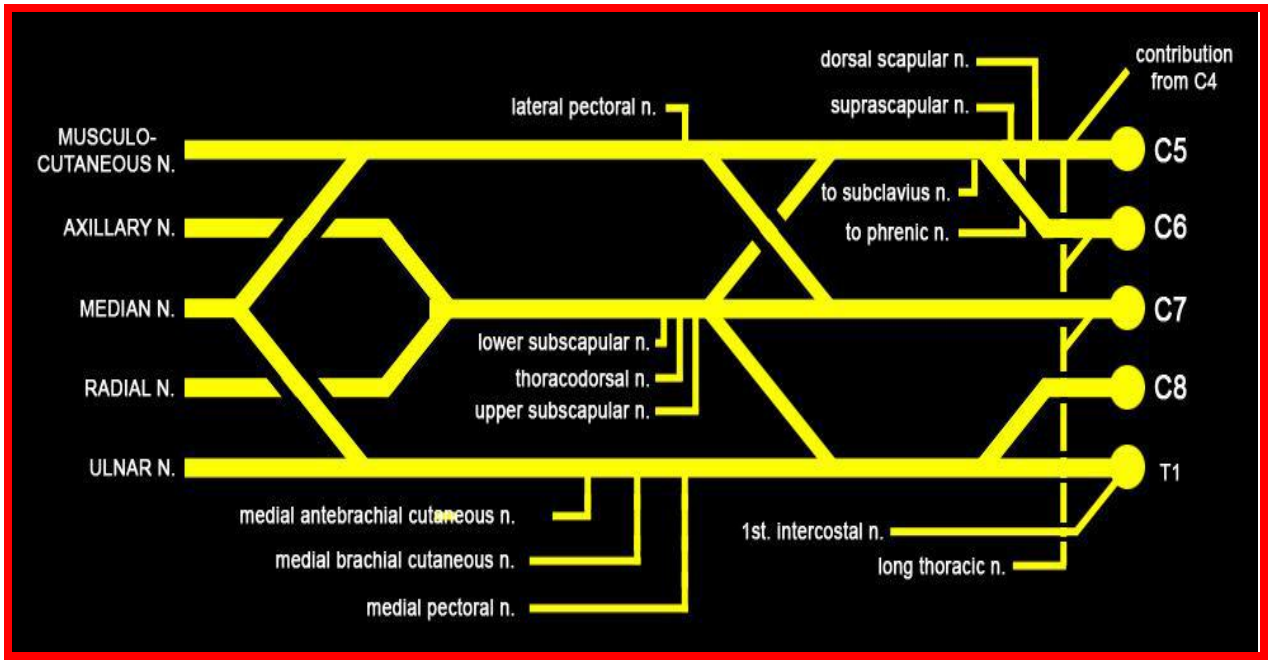

Fig.4 Brachial Plexus and its Terminal Branches

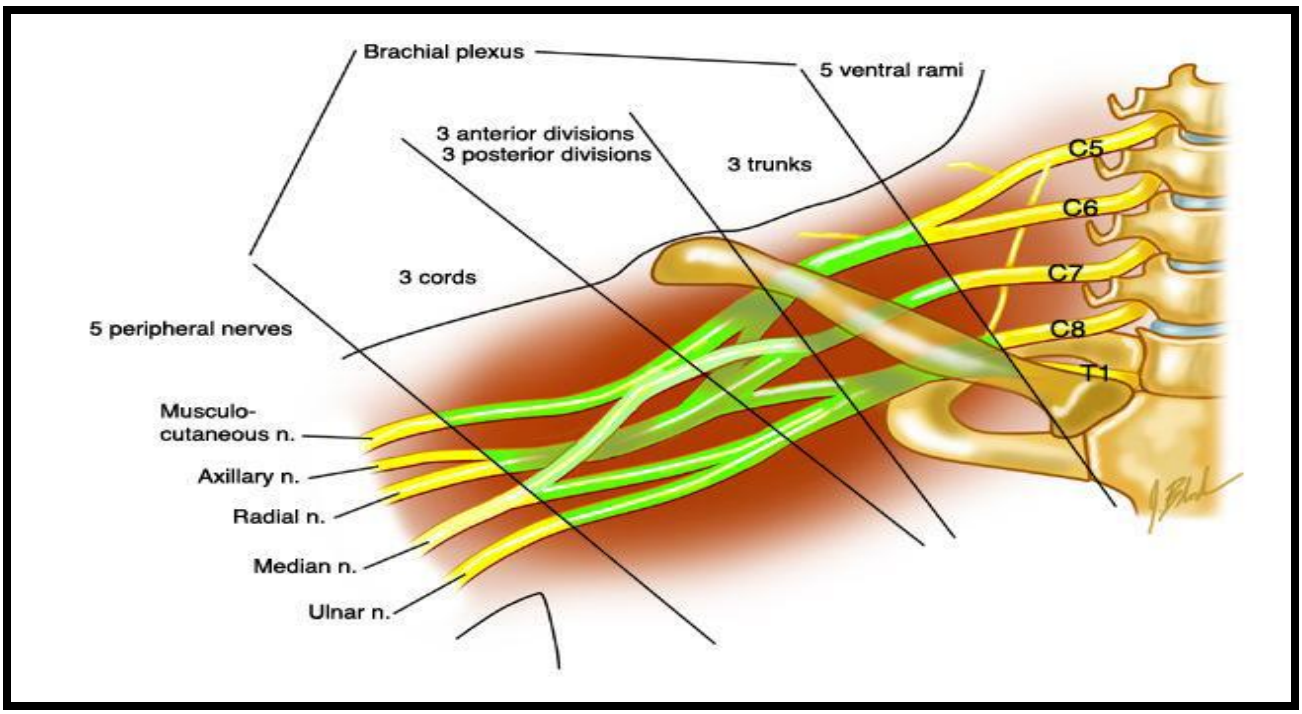

Fig.5 Dermatomal Distribution of Upper Limb

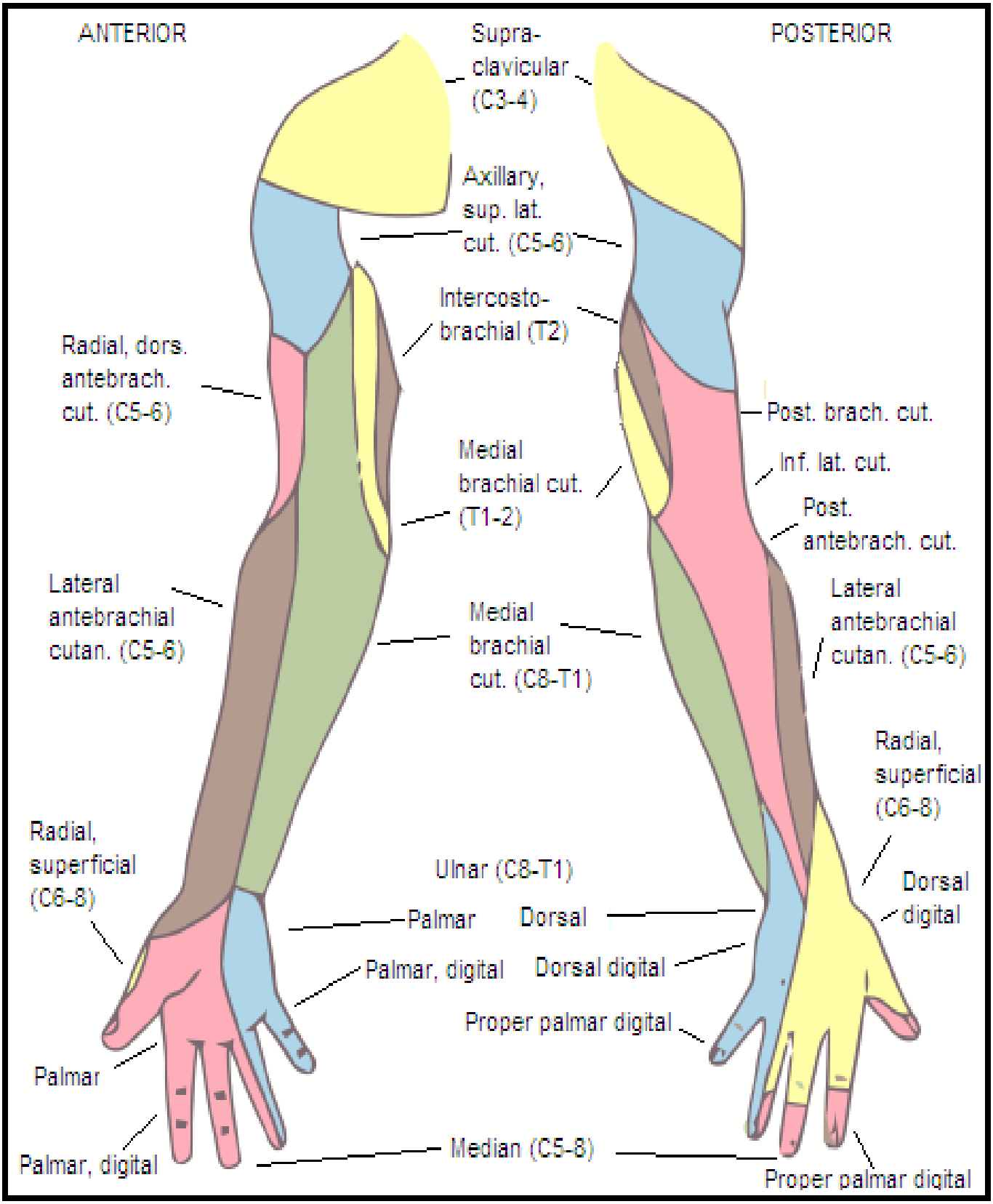

Fig.6 Pain Pathway

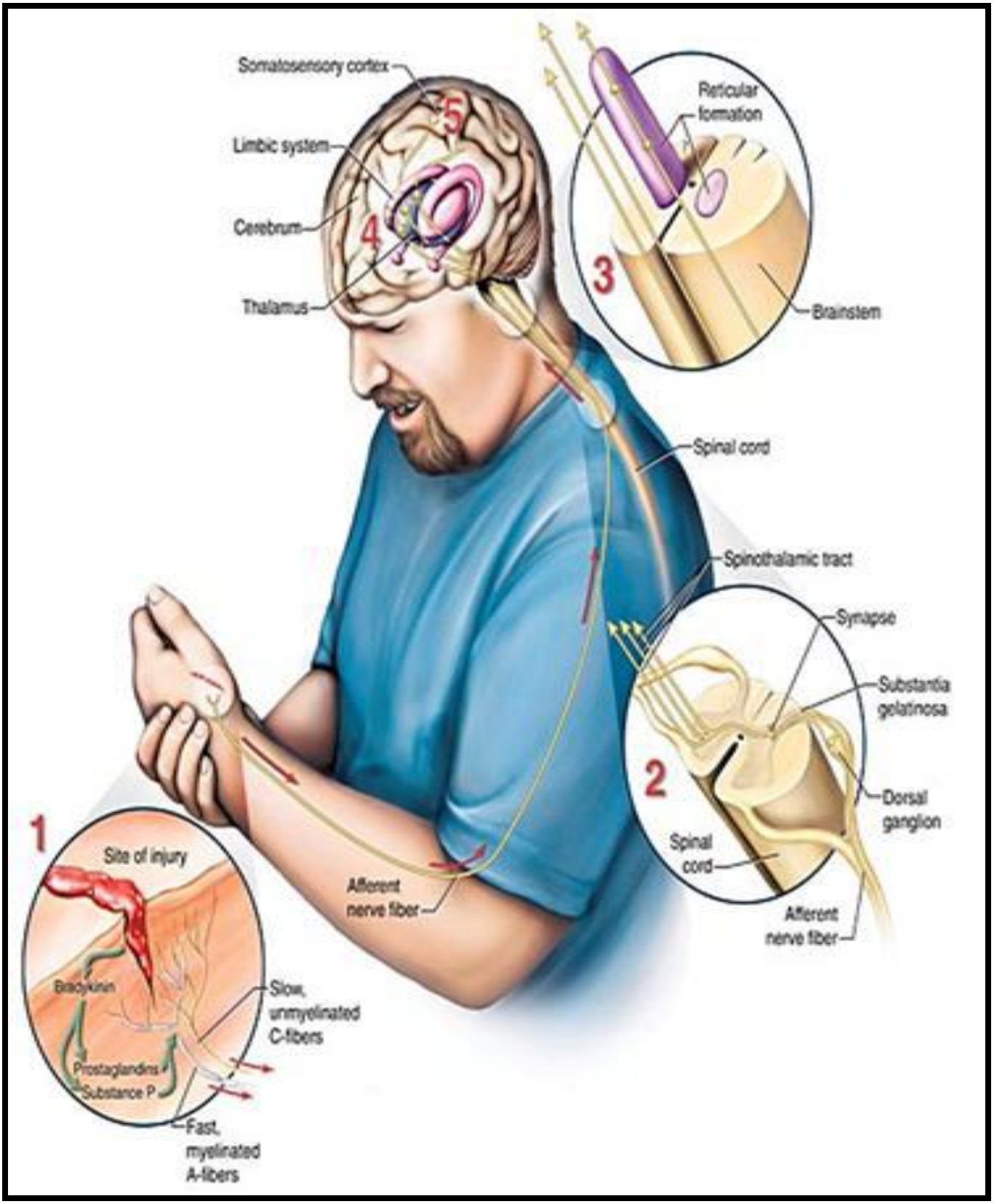

Fig.27 First brachial plexus anaesthesia at 1885.

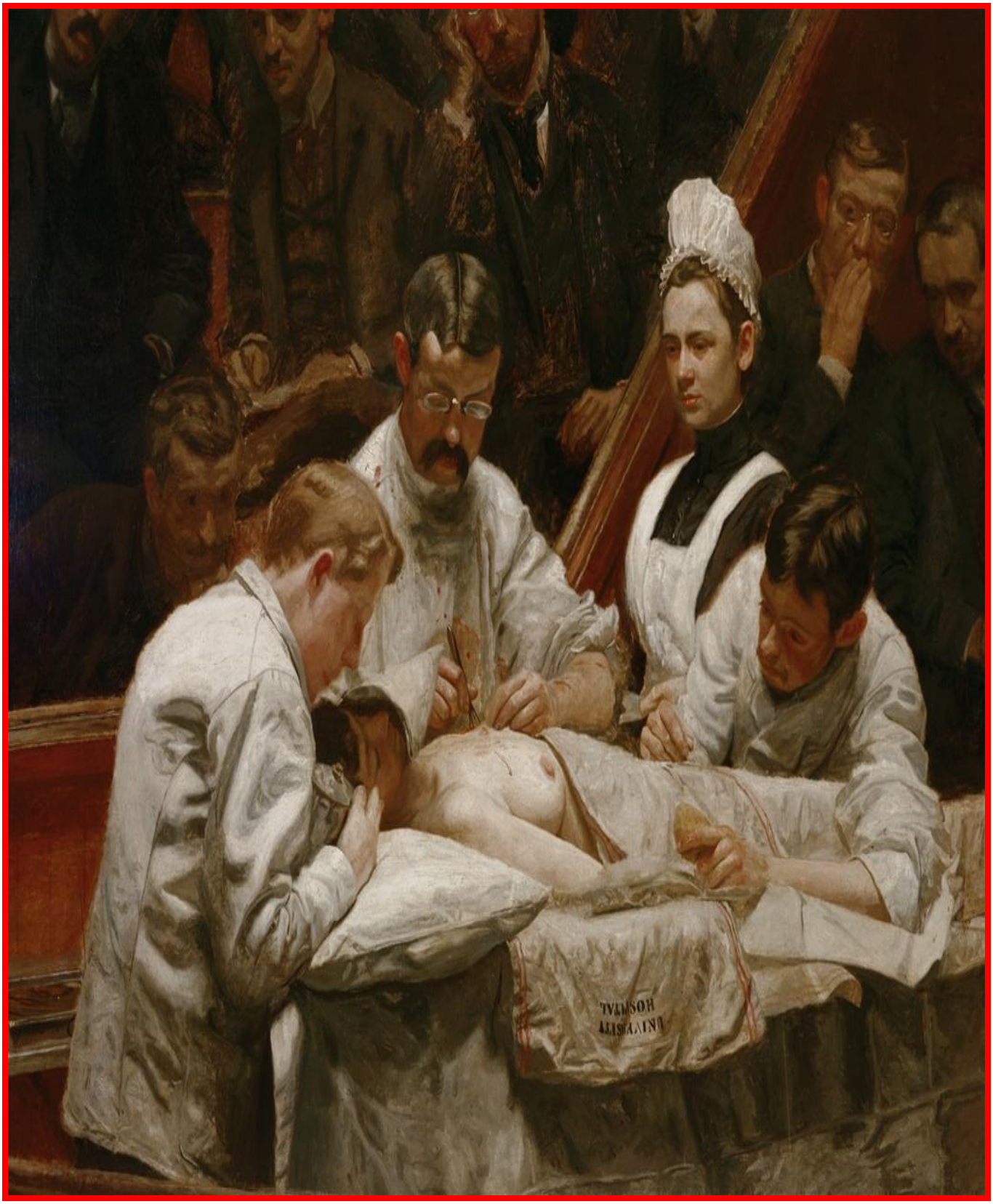

Fig.9 Ultrasound Signals

Reflection from structures generates an ultrasound signal. A structure perpendicular to the beam will generate maximal reflection. A nerve or a needle at a steep angle will cause less reflection of sound waves to the probe.

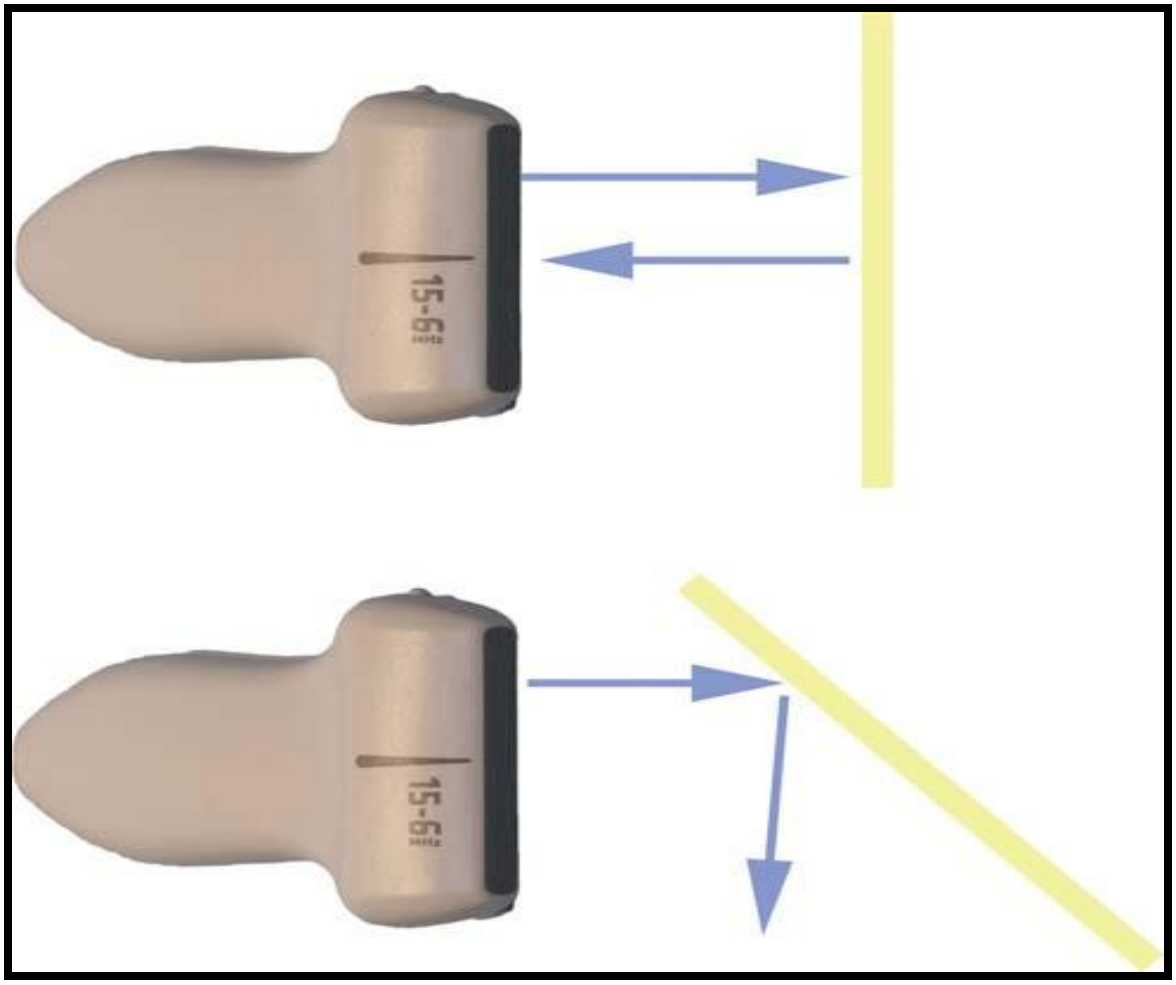

Fig.10 Different types of probes

A large, high-frequency linear probe (left); a small linear probe (middle); and a large, low-frequency curvilinear probe (right).

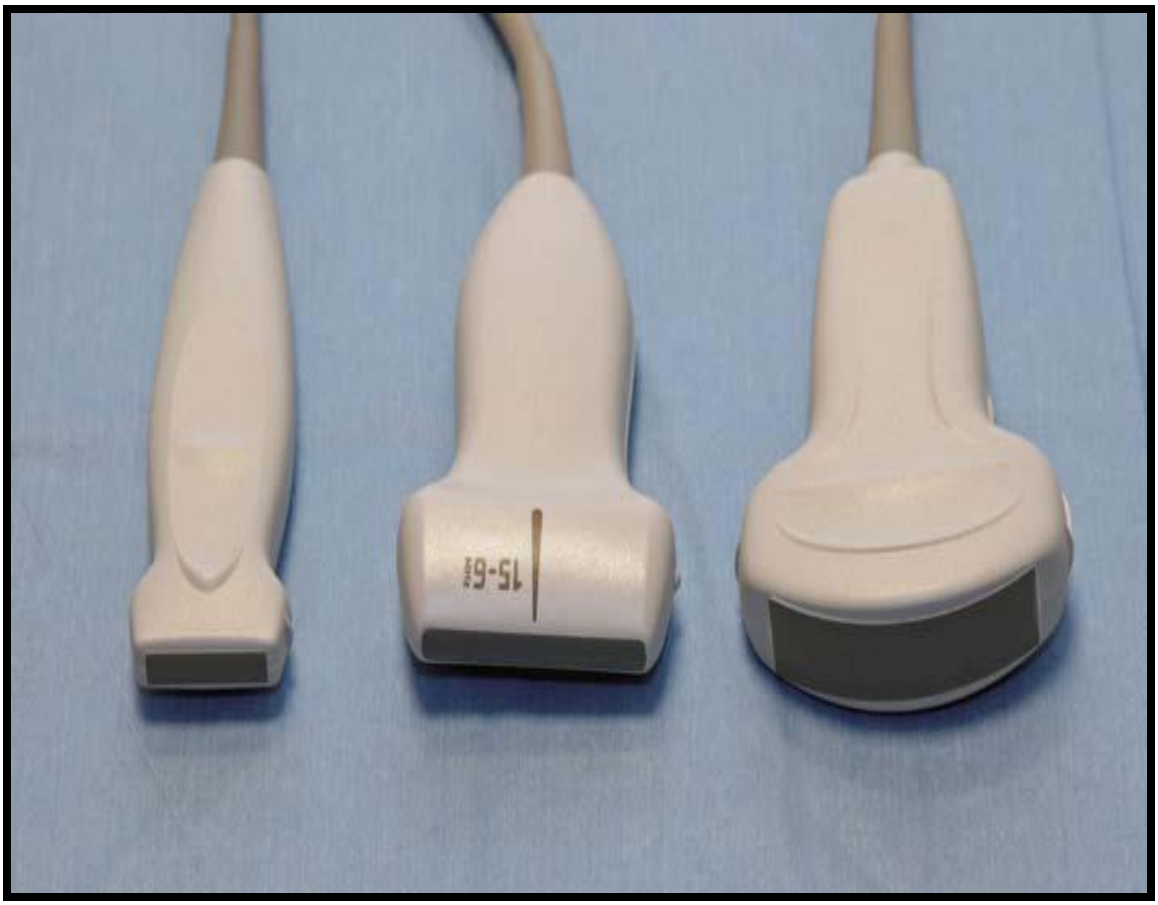

Fig.11 Using the highest frequency

To achieve the best axial (vertical) resolution, use the highest frequency possible. This reduces the wavelength of the sound waves and makes discrimination of small structures possible. The tradeoff is poor tissue penetration. The short wavelength/high-frequency sound waves dissipate more energy, leading to less tissue penetration.

Fig.12 Focus position affects image quality.

The identical interscalene anatomy is scanned on 2 images. On the left the focus is set deep and on the right the focus is set shallow. The nerves are easier to identify on the right, where the focus is set at the same depth as the nerve roots in the interscalene groove.

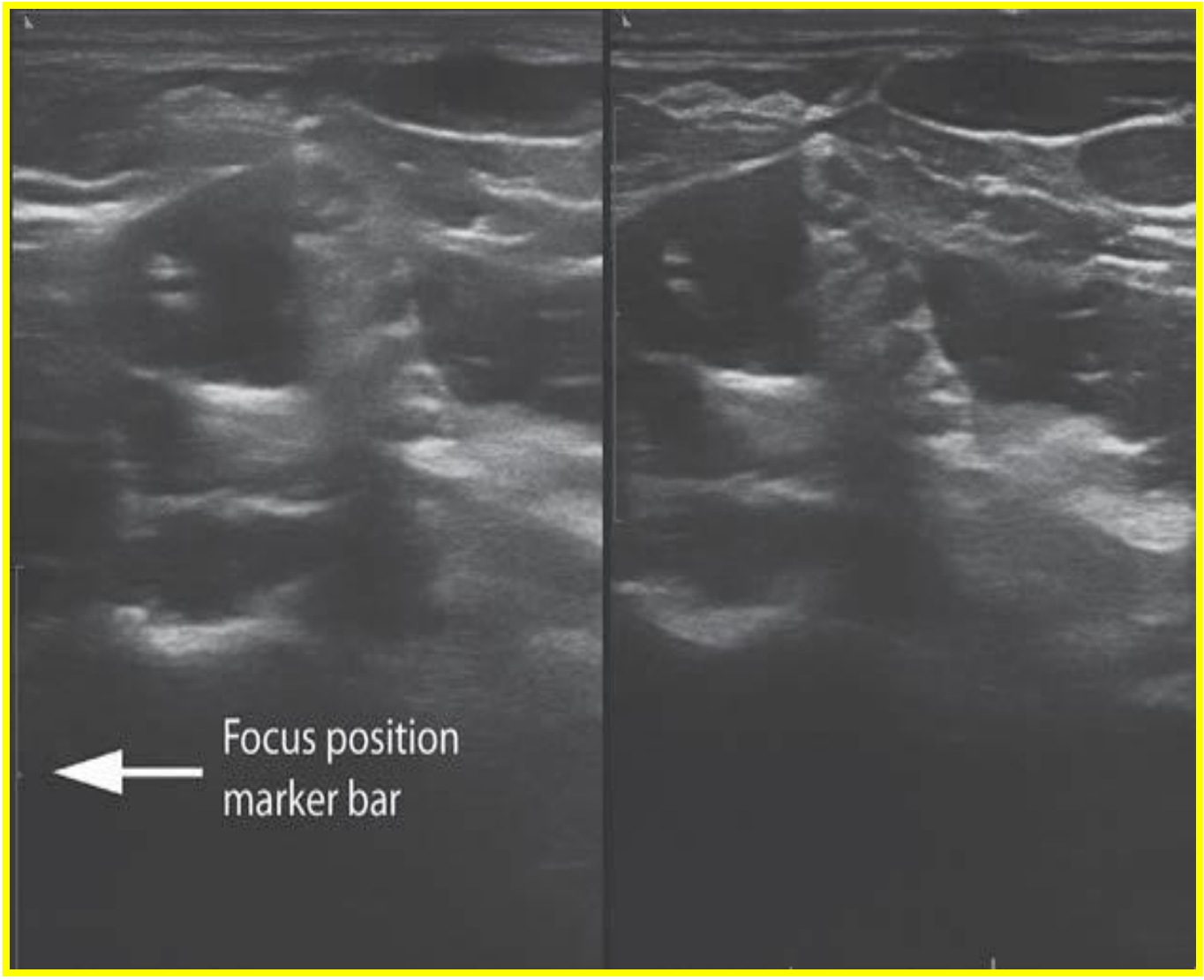

Fig.15 Color Doppler can help identify vessels.

Tilting the probe can make vessels appear to have flow or no flow. This is important to help discriminate vessels from nerves. The images are of the same artery with the probe at anacute angle the upper image and the probe perpendicular to the vessel in the lower image

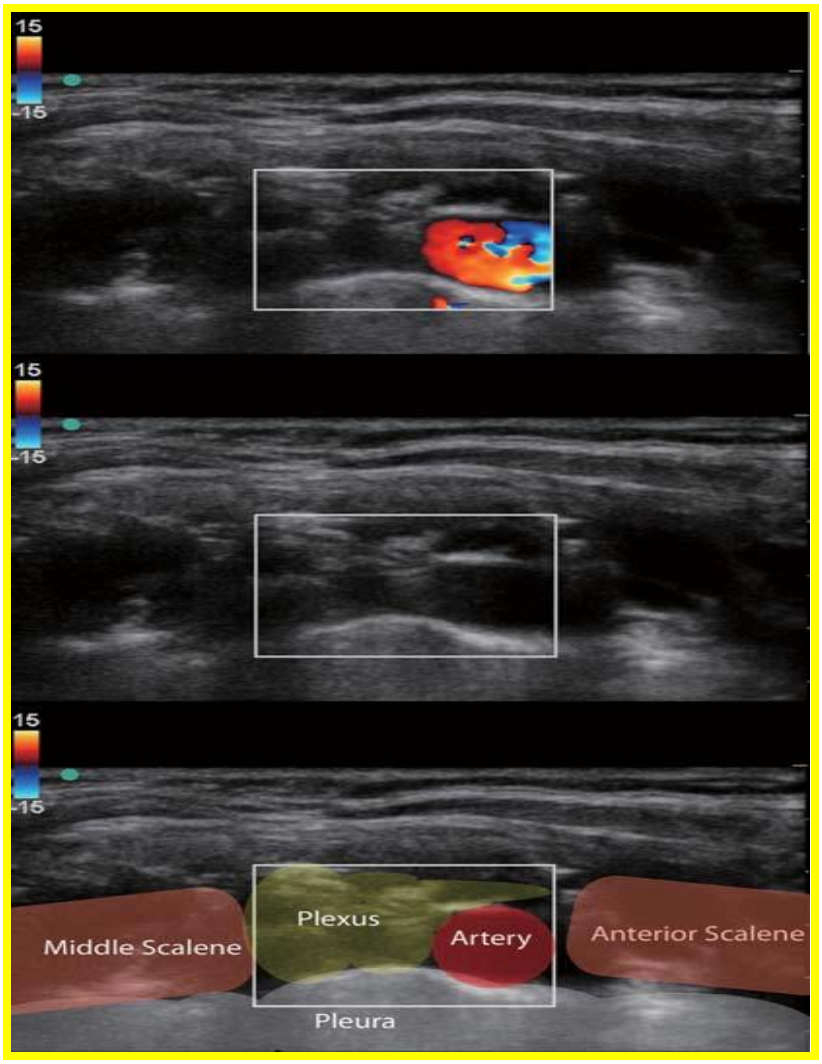

Fig.13 Over- and Under-Gained Images.

The center image demonstrates the bright radial nerve in the center. The under-gained image of the same nerve above is very dark, and the over-gained image below is very bright. Both over- and under-gain lose vital detail.

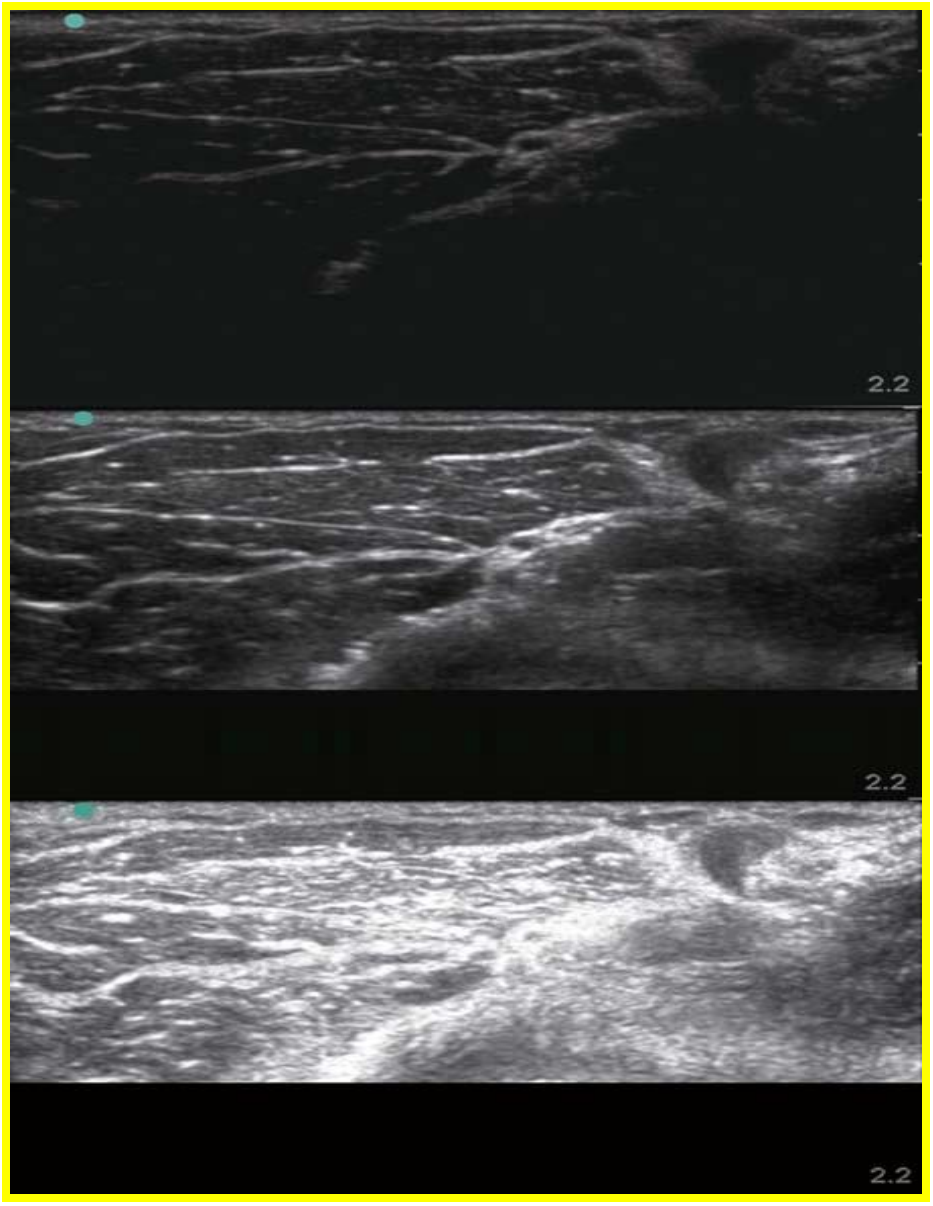

Fig.14 Time gain compensation (TGC)

It adjusts brightness at various depths of the screen. This can lead to artifact if not set correctly. On the left the TGC is set appropriately and the nerves are visible. On the right one of the nerve roots is not visible because the TGC bar has been moved across.

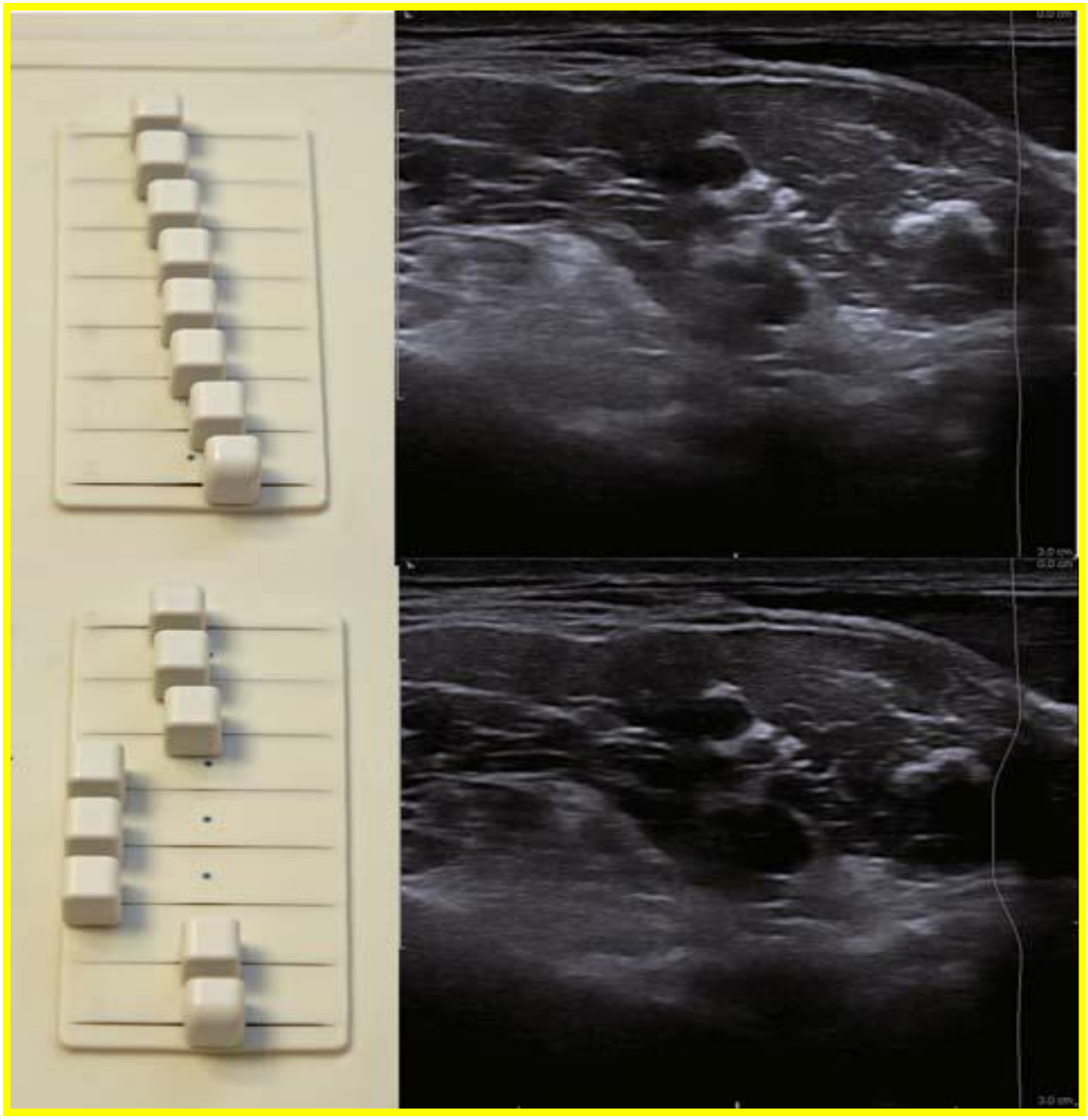

Fig.16 In-plane & Out-of-plane Needle Approach

The upper images demonstrate an in-plane needle approach. The lower images demonstrate an out-of-plane needle approach.

Fig. 17 Out-of-plane Technique - Sliding.

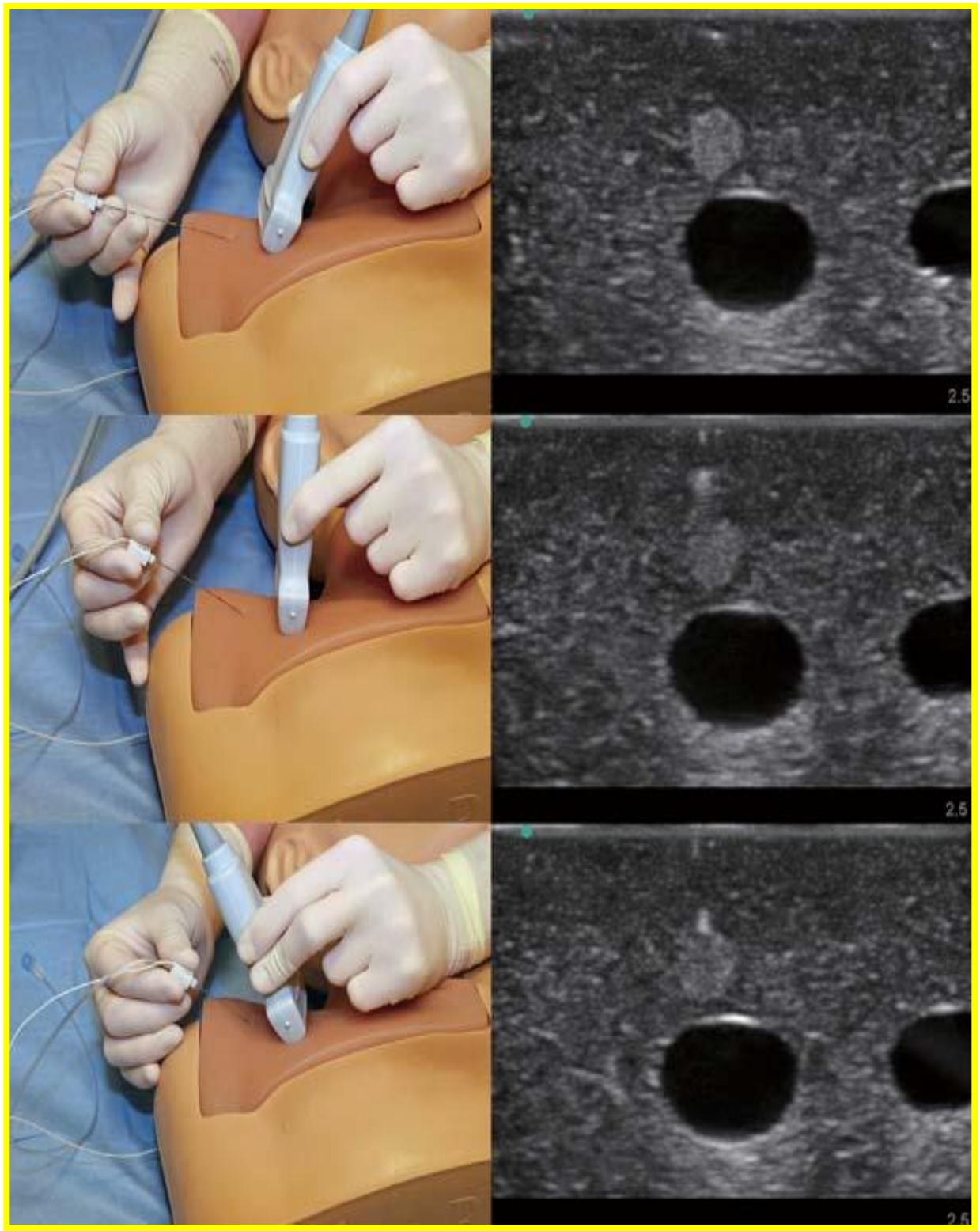

Fig.18 Out-of-Plane Technique - Tilting.

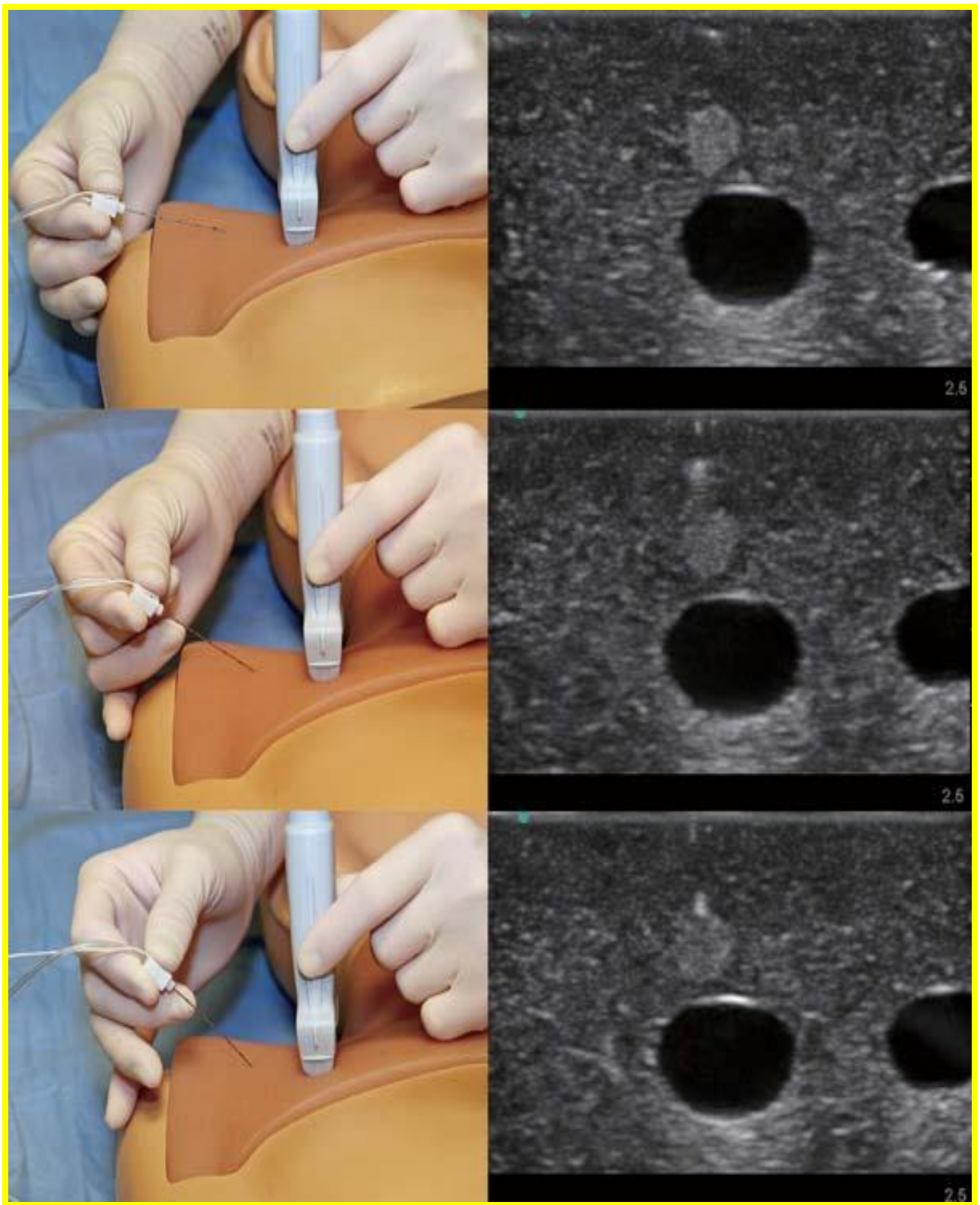

Fig.19 Out-of-Plane Technique - Adjust the Needle.

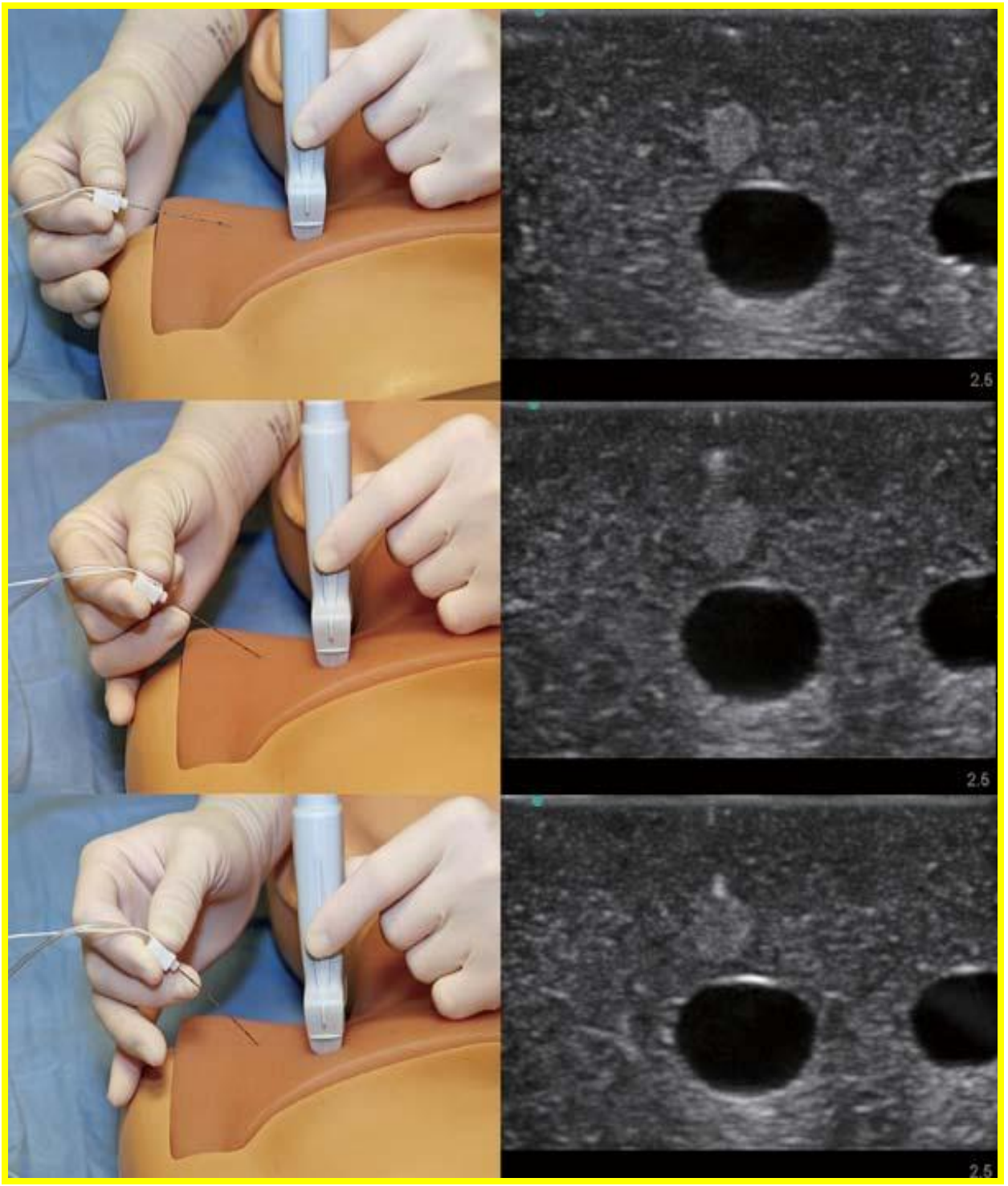

Fig. 20 Dropout artifact is caused because the entire foot of the probe is not in contact with the skin surface.

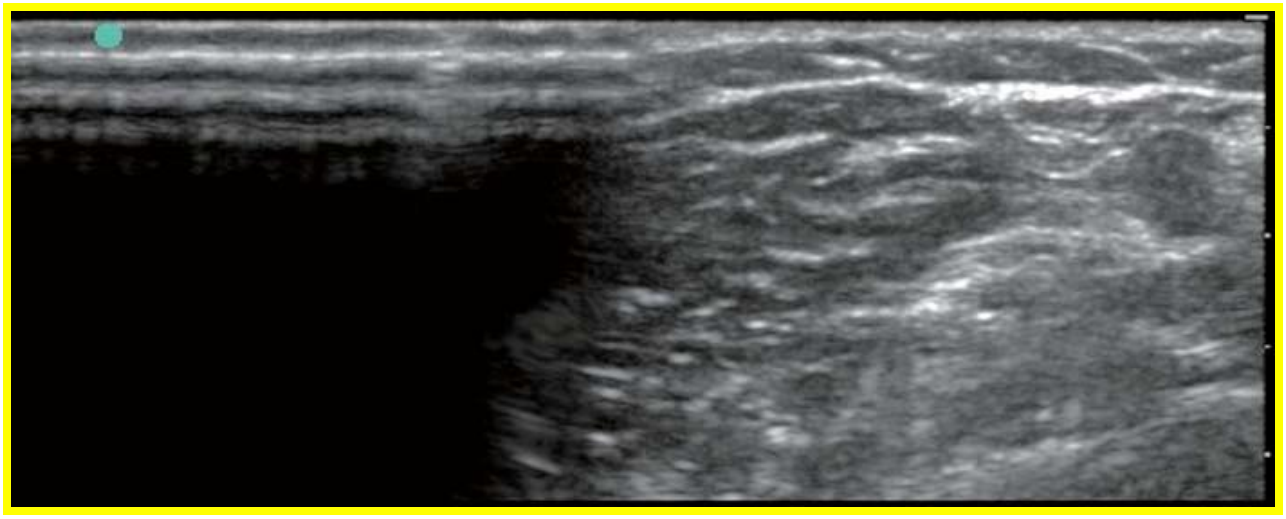

Fig. 21 Attenuation occurs as sound waves penetrate tissues. On the left the image is dark, and on the right the distal gain has been increased to make the distal portion of the image more visible. Other techniques to view deeper structures are reducing the frequency and adjusting the focus to a deeper position.

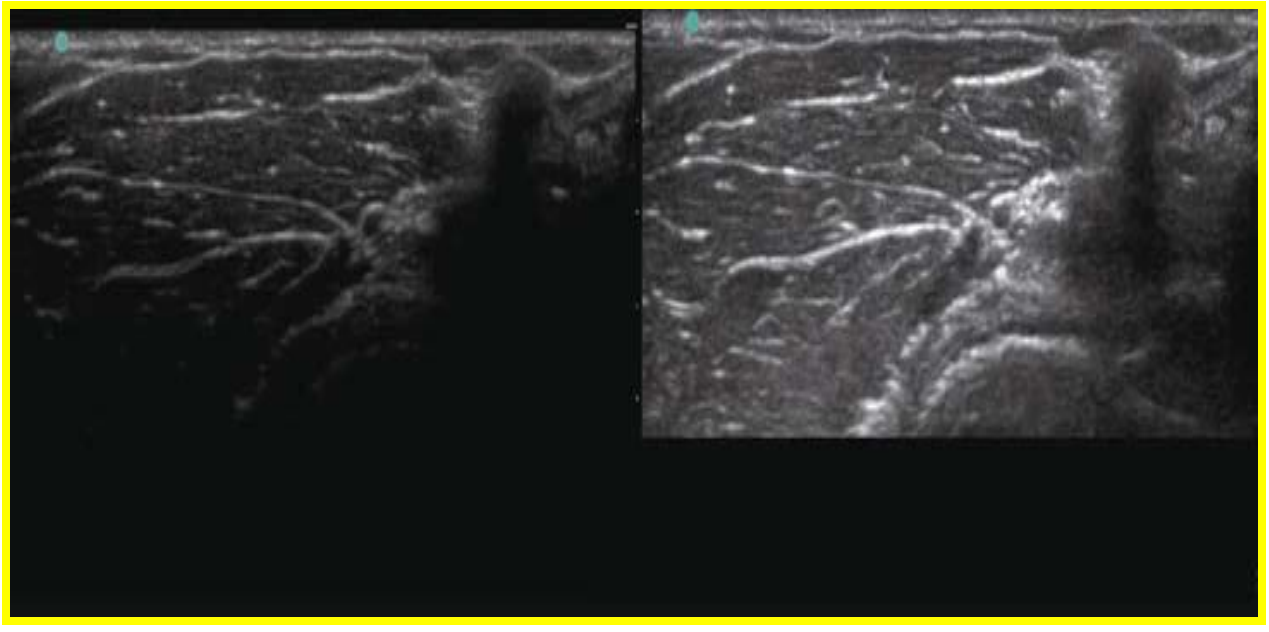

Fig. 22 Needle misalignment is common and can lead to incorrect identification of the needle tip. In the two ultrasound images the needle has not moved, but because of misalignment the needle tip position appears different.

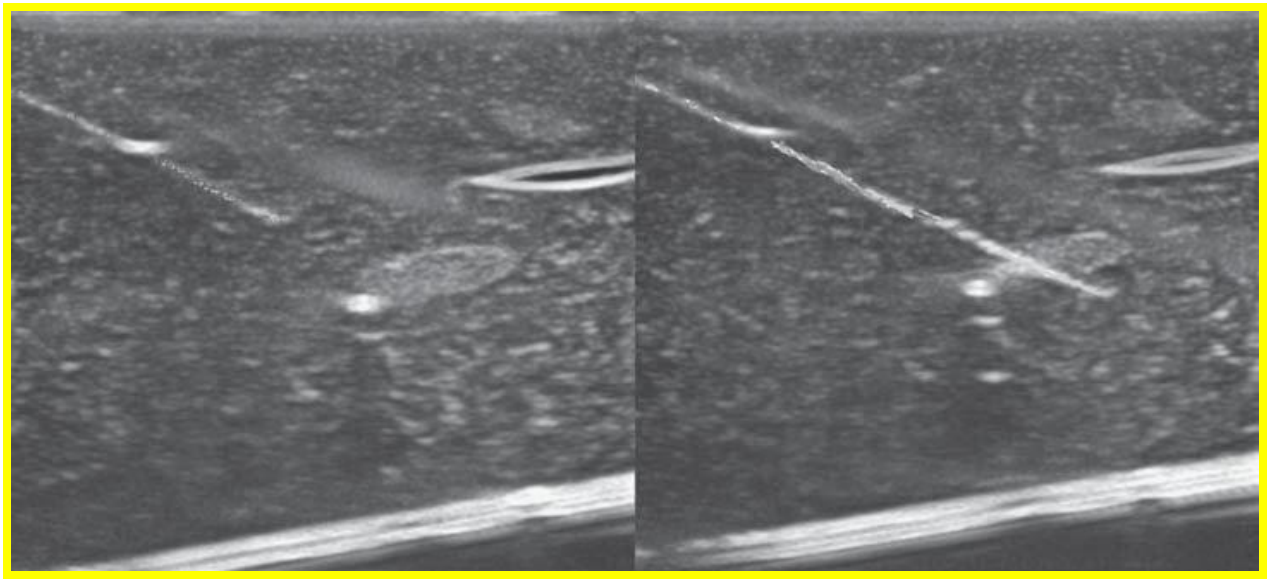

Fig.23 Bayonet effect.

The needle appears to bend as it passes through muscle and into the local anesthetic pool.

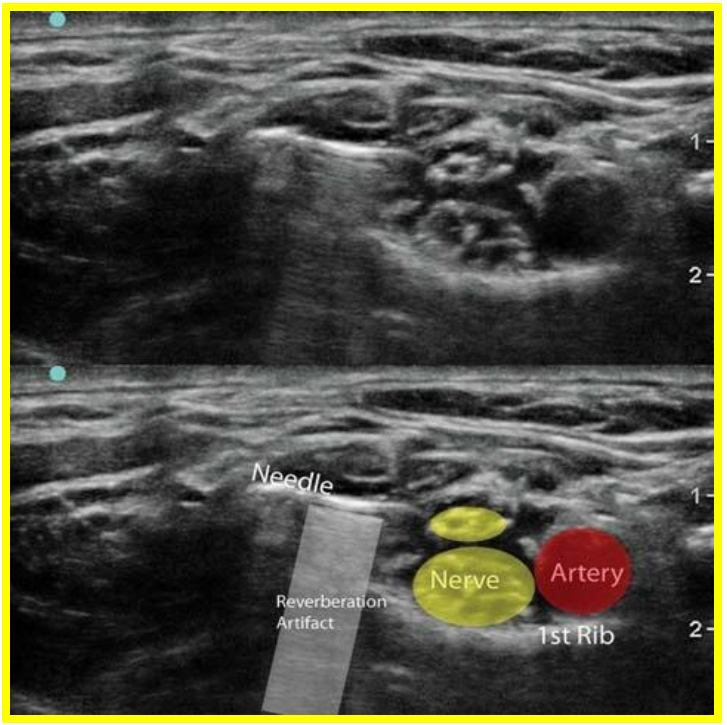

Fig.24 Reverberation artifacts.

In this interscalene nerve block the reverberation artifact beneath the needle obscures the image of the interscalene groove and nerve roots beneath the needle tip

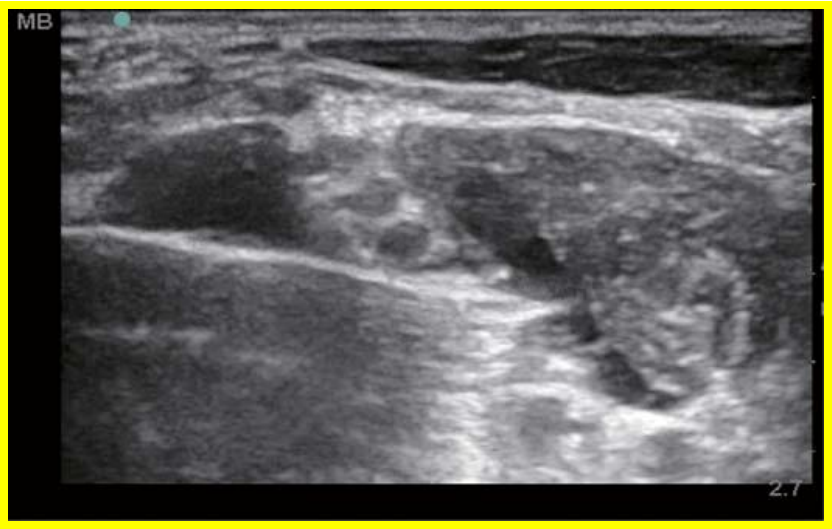

Fig.25 Intraneural injection.

The upper left and right pictures demonstrate before and after an intraneural injection within the musculocutaneous nerve (MC). In the lower two identical pictures the nerve has been highlighted yellow.

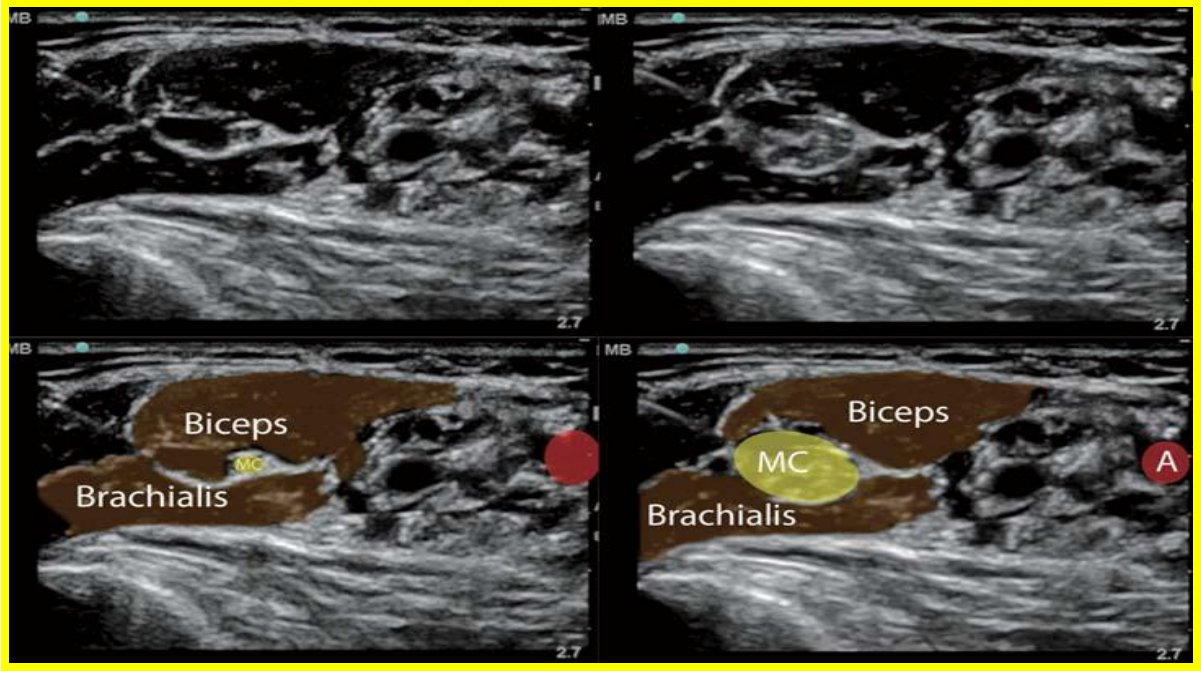

Fig.26 Equipment needed for an auxillary block